BRAIN.

PART II., 1890

Original Articles.

THE PSYCHO-PHYSICAL PROCESS IN ATTENTION.[1]

BY J. SULLY.

Read before the Neurological Society on December 19, 1889.

I PROPOSE to discuss a subject that seems at first sight of more consequence for the pure psychologist than for the neurologist. I hope, however, that I may succeed in showing that the topic has its importance for all who make a study of psychical phenomena: whether, as psychologists, they are primarily interested in these phenomena on their own account, or, as neurologists, they deal with them rather as concomitants and symptoms of those nervous processes with which they are more immediately concerned.

I may probably assume here that we are agreed upon the possibility of a true science of psychology, or, if the expression is preferred, physiological psychology, which proceeds by bringing together as closely as possible the results of subjective and objective research. And here I trust I may not seem presuming if I remark that in this work of co-ordinating the two kinds of knowledge—that of our conscious states and that of the functional activities of the nervous system—there must be on either side a certain give and take. On the one side the psychologist should welcome every new discovery that helps to elucidate the obscure and subtle actions of nerve and nervous centres. On the other side, he may rightly claim that a certain respect be paid by the neurologist to the results of psychological observation and

[1] It is hoped that a discussion of the subject of the paper will appear in the next number.

analysis. We must remember that in this conjoint investigation of psycho-physical processes from the subjective and the objective side, the psychologist has had the advantage of a start. With respect to many of the more complex phenomena involved, every candid neurologist will, I believe, admit that the knowledge of the physiological factor is meagre and mostly conjectural. And it is precisely here that the psychologist claims to have done something in the way of simplifying by analysis the tangle of complexity. Not that the candid psychologist—for I trust that there are such to be found—would contend that his results are above suspicion. Every fair-minded student of psychology will at once grant that the history of the so-called science presents much more a congeries of individual systems than one organised body of verified knowledge. All that he will ask, if he is reasonable, is, that while he is gratefully accepting the fruits of neurological research and seeking to adjust them to known mental processes and laws, the neurologist should pay him the reciprocal courtesy of pursuing his researches in the light of the best and most assured results of psychological analysis.

These remarks seemed to me almost necessary in introducing to a gathering of neurologists the subject of Attention. Attention, in its higher form of voluntary selection and fixation of particular presentations of sense and ideas, is apt to be thought of as a mysterious emanation of spiritual activity that interferes with and controls the mechanism of mind. According to this view, it is the occult *Ego* that asserts itself in the activity of attention, and by scanning the given contents of the moment, and by separating and re-arranging these, is itself the author and creator of its higher intellectual life. Attention, in fact, has been involved in all the psychological mysticism which has clouded the processes of volition. The spiritualist in psychology who fights hard for the spontaneity and liberty of the subject against the upholders of mechanical uniformity, naturally betakes himself to the lofty region of voluntary attention as an inexpugnable akropolis. And even some writers on the physiology of mind have treated the whole process of

selective attention as standing outside the circuit of psycho-physical processes—an alien presence, "mystic, wonderful." As a natural reaction against this conception of attention as a recurring miracle, the physiologist has, I suspect, been disposed to ignore it, or, at least, to under-estimate the large part played by it in the mental life. He may not have gone so far as Condillac, who defined attention as nothing but the transformation which a sensation undergoes when it predominates by its vivacity. Yet he has, I think, tended to overlook its distinguishing characteristics and its common co-operation in our psychical processes. "Sensations (he has seemed to say) I know, and movements I know; but who are you?"

But a *rapprochement* of the psychologist and the neurologist is recognisable here also. The latter has come to recognise that our sensations, in their vividness and persistence, are determined not only by conditions in the peripheral process of stimulation, but by a reflex central reaction. This, indeed, naturally follows from the conception which seems to be now commonly adopted, that every psycho-physical process is at once sensory and motor. A good deal has been done by Fechner, Bain, Wundt, Ferrier, and others towards determining the precise nature of this nervous reaction. The claims of attention, moreover, have not been lost sight of in assigning special cortical seats to the several distinguishable psychical claimants. In England, as well as in Germany, the question of the precise region of the cortex involved in the process of attention has been the subject of considerable discussion.

In view of this hopeful state of things, I venture this evening to urge the claims of the subject on neurologists. I need hardly add that I shall endeavour to conceive of attention as a process at once psychical and physical, of which we may, by properly scientific methods, determine the co-operant factors and conditions.

I propose to give at the outset a brief *résumé* of some of the more important results of the psychological treatment of the subject; then to see what recent experiment has done to make these results more complete and exact; after this, to

consider the current views respecting the nervous process involved; and, lastly, to point out some of the practical bearings of the subject, and more particularly its relation to healthy brain-action and to normal life.

(A) *Psychology of Attention.*

I must here confine myself to a few of the more important points.

In the first place, then, I think it may be said that recent psychological research has tended to the conclusion that the common and typical form of psychosis is complex in which a presentative element of sense, or its ideational equivalent, is followed and reinforced by a distinctively active element of attention—a third element of feeling, that is pleasure or pain, being commonly, if not in all cases, interposed between them. According to this view the simplest fact of consciousness, when it presents itself in a complete and distinct form, is determined in part by a mental reaction. In many cases the two factors—sensation and reaction—are easily distinguishable, as when attention follows slowly on the persistence of a sub-conscious sensation, as, for example, in listening to the repeated stroke of the clock or other prolonged sound, or in directing attention to some obscure organic sensation; or when, on the contrary, the direction of attention precedes the occurrence of the sensation as a process of mental preadjustment or expectant attention. In other cases it is less easy to separate the two constituents; for instance, when surprised by a loud sudden sound, or mastered by a teasing and persistent idea. Closer inspection will however show in these cases, too, that the completed result involves a reaction. A sudden sound is not distinctly heard for an appreciable interval, during which the reactive process presumably adjusts itself. And in the familiar experience of going back unwillingly at night to some worry of the day, it is easy to see that not only do the ideas recur, but that after a vain struggle to exclude them perhaps we proceed to react in the customary way, just as if it was of the first importance for us to settle the perplexing point at this very moment.

The psychologist distinguishes this reaction as having the characteristics of an *active* phase of consciousness. This he does solely on the ground of its qualitative peculiarities, and without any *arrière-pensée* of an active spiritual principle. Attention is an active phenomenon, in precisely the same sense as conscious movement is active. It presents in its several variations all the known active elements, as craving, effort, attainment. This active phase becomes most distinct in those more complex forms of the process commonly marked off as voluntary or volitional attention, where there is a keen sense of resistance and struggle—as when by a special effort a man attends to a matter when suffering great bodily discomfort, or plagued by some disturbing noise.

The consequence and evidence of the completed process of attention or active mental adjustment is the fixation of the particular sensation. Attention is Detention in consciousness.[1] This detention instantly brings about or is attended by a certain rise in vividness or intensity, and also by a very considerable increase in distinctness. When I attend, for example, under favourable circumstances to a sound, I make it for the time the predominant mental content. I may just add in passing that the rise in intensity and in definiteness together, due to attention, differs from the rise in intensity due to an external cause in two particulars: (*a*) The rise in intensity is narrowly limited; (*b*) it subserves and seems subordinated to the increase in definition. A musical sound of a given pitch does not go on becoming more definite in quality as it gets louder; but, on the contrary, after a certain moderate intensity, becomes less definite. But attention uniformly tends to definiteness of quality.

Lastly, psychology tells us that attention has some at least of its conditions among conscious phenomena. The simplest supposition would be of a sensation calling forth a reflex attention in the absence of all contending forces. This idea is of course never realised. At no moment is our

[1] This is clearly illustrated by Prof. Ribot in his recent volume, *La Psychologie de l'Attention.*

consciousness reduced to a single presentative element. But there may be an approximation to this state of things, as when surprised by a noise in the silence of the night. In such a case we say the reaction is the direct result of the sensation itself. Among the features of sensation which have most to do in determining this reflex attention may be named intensity, suddenness, or unexpectedness, also duration, including recurrence, as in the case of the clock-strokes. The effect of suddenness, as mental shock, is familiar. A sound, if gradually introduced, might fail to call forth attention, even when attaining a considerable intensity. It may be remarked further that all increase in intensity does not further the reactive process. A very loud sound "overpowers us," as we phrase it—that is, baffles the adjustive process of attention.

The first step in the complication of conditions is where the sensation is distinctly pleasurable or painful. Here attention is prompted by feeling, and begins to follow the directions of interest. I here assume that both pleasure and pain have at first the effect of rousing attention. This is, I think, borne out by observations of familiar facts, as children's and grown-up people's susceptibility to the fascination of the horrible, and the vast capacity of most people to make themselves supremely unhappy. There seem to be good reasons, too, from a biological point of view, why we should attend to painful sensations, which in most cases at least indicate a harmful action on the organism. The preferential attention to pleasurable sensations is, I conceive, an acquisition involving volitional selection.

The next stage of complication is the partial determination of the direction of attention by pre-existent lines of interest, involving the residua of previous experience, and habitual forms of mental activity. That as we grow older all the more vigorous sort of attention tends to confine itself within certain limits answering to our special pursuits, information, and tastes, is a trite observation. We each see what we are prepared, by the special circumstances of our life, to assimilate or understand, feel the worth of, or turn to practical account. This cumulative effect of the past on the

present in fixing predominant lines of interest has not, I think, been adequately dealt with by English psychologists. The Herbartians in Germany treat of this under the head of that much-abused word Apperception.

The last stage in the complication of the process, is what psychologists mark off as voluntary or consciously selective attention. Here a number of forces is supposed to be at work, and attention is prompted hither and thither. There emerges the complex psychical phenomenon, a conscious resolve to attend to one particular sensation or idea under the stress of a predominant desire or motive. This volitional process has for its distinguishing accompaniment what is commonly spoken of as mental effort, and sense of resistance.

(B) *Recent Psycho-physical Experiments.*

Our knowledge of the process of attention, its variations, and its effects, has been materially increased by recent psychological and psycho-physical experiment. Attention is a subject on which the psychologist may expect the physiologist and the scientific worker in general to throw some light. Observation, as we all know, implies attention in its highest and most disciplined form, as steady, prolonged, mental concentration on the phenomena to be recorded. Hence it is not surprising that valuable psychological facts relating to the working of attention have been contributed by physicists, more particularly, observers of optical phenomena. I may refer to the apparent changes in the appearance of optical phenomena due to variations of attention, as the transformation of relief in a drawing, according as the observer attends to the one or to the other aspect, and the quenching of one coloured field by another, when the observer fixes his attention on this last.

It is, however, in a long series of experimental inquiries into what is known as reaction-time that the effects of attention have been most fully elucidated. There is no need to attempt any detailed account of these here, as they have already formed the subject of a paper contributed to BRAIN.

I will therefore content myself with a reference to one or two of their main results.

One of the most valuable results brought out by these experiments, is, I think, the fact that attention to an impression of sense is a process of adjustment which occupies an appreciable and measureable time, this time being longer or shorter according as the conditions of adjustment are unfavourable or favourable. Thus to take an unfavourable case, it is found that the reaction-time is appreciably lengthened when a disturbing cause, *e.g.*, an organ playing in the same room is at work; in the case of a person whose normal reaction-time was 100σ (where σ is $\frac{1}{1000}$th part of a second), it rose under these circumstances to 148σ. If the subject again is taken completely off his guard it may go up to 500σ (half a second). On the other hand, if the process of adjustment is carried out to some extent beforehand, the reaction-time is reduced. For instance if the subject knows by some signal the instant at about which the impression is to arrive, the reaction-time may fall from 253 to 76σ. When the preadjustment is complete an illusory perception with respect to the exact moment of the occurrence of the impression may arise, and the subject react too soon.

These experiments have been elaborated and varied by Wundt and his pupils in different ways. Thus, instead of a simple, sensory stimulus, on which the same reaction—say a movement of the hand—uniformly follows, one of a number of stimuli is applied, and the subject required to carry out one of a number of movements, *e.g.*, of the different fingers corresponding, by previous arrangement, to the particular stimulus selected by the experimenter. In this way Wundt considers that he has been able to determine the duration of different psycho-physical factors in complex processes, such as discrimination time and volition time.

These experiments, interesting as they are, have not yielded such certain results as one could wish, for Wundt's own interpretation of the facts reached is not quite satisfactory, owing to the language he employs in describing the process of attention, or as he calls it, apperception. His

whole theory of apperception has been severely criticised by Dr. Bain, and more recently by Dr. Hugo Münsterberg in a very remarkable publication, *Beiträge zur Experimentellen Psychologie*. Münsterberg has worked out a series of experiments himself and claims to have demonstrated that complex process involving rudimentary acts of judgment can be carried out under certain conditions in a purely mechanical way, and without the intervention of Wundt's indeterminate element, the selection of a particular impression by a volitional act of attention, and of the corresponding movement by a volitional impulse. Dr. Münsterberg effects this by getting the subject of the experiment, after a careful preliminary association of the several kinds of sensory signals (here words), and the corresponding finger-movements, to divert the attention altogether from the expected sensory signal and to concentrate it on the movements. Under these circumstances he finds that the reaction-time does not increase with the complication of the process as Wundt contends, but remains approximately constant. In these cases, then, there seems, as Münsterberg argues, no room for the discriminative and the volitional periods. The whole process becomes indeed largely unconscious or sub-conscious.

I have referred to this latest outcome of psychological experiment in Germany because it seems to me to illustrate in a new and striking manner the effect of pre-adjustment. The possibility of the whole process lapsing, in this instance into a quasi-unconscious one, is plainly due to the circumstance that the work of attention has been done in advance. Thus the subject passes, say, from the sound of the Latin word lup-*o* to the movement of the particular finger agreed upon as the digit which is to respond to the dative case, because in the preliminary mental fixation of the dative case and the associating of this with that particular finger-movement, the sound of the inflexion *o* was included. Similarly in the case where the signal "Schiller!" is instantaneously followed by the particular movement which has been already co-ordinated by a severe preliminary effort of mental concentration with the sound of the name "poet."

(C) *The Nervous Process in Attention.*

We may now pass on to consider the exact nature of the physiological process in attention. That attention stands in a particularly close relation to the process of motor innervation is a fact that discloses itself to even superficial observation. If we take the simplest form of attention, that directed outward to an impression of the sense, it becomes obvious that the mental adjustment is effected, in part at least, by a muscular adjustment. Thus, in the case of expectant attention, when we are awaiting an impression of sight or of hearing, it is easy to recognise in the pose of the body, the movement of the head and eyes, the presence of a vigorous motor factor. This factor does not manifest itself exclusively in actual movement. One would rather say that the characteristic of this muscular exertion is fixation of body, limb, and sense-organ, by a balanced action of antagonist muscles, and by the inhibition generally of all diffused movement.

In the case of fixating ideas the motor factor is less patent than in that of sensuous attention; but it is nevertheless present and observable. Fechner was one of the first to give a careful account of the muscular sensations localised in the head which accompany the effort of thinking. Others, as G. H. Lewes, have layed stress on the part played by the respiratory muscles in the severer acts of intellectual concentration. Dr. Bain goes farther, and seeks to account for the whole process of thought-control by help of a motor process. According to him, every idea is compounded of an element of passive and of muscular sensation. When, by an effort of will, we fixate or transfix the idea, it is because we can by the proper mechanism of volition, the motor apparatus, act upon the muscular factor—that is, I presume, strengthen and intensify it by a complete or partial renewal of the original movement.

In considering the sufficiency of the hypothesis that attention is a motor phenomenon, pure and simple, two points need to be examined: (1) Does it harmonise with what we know subjectively respecting attention? (2) Does it account for the known effects of attention?

On the first of these points I have little to say. That there is a close affinity between muscular and mental exertion I readily grant. Each is a variety of the active phase of consciousness. This affinity is illustrated by the fact that psychologists of eminence as Wundt in Germany, and Ward in England, regard both modes of activity as at bottom one.

And here may I be allowed to say, in passing, that from a psychological point of view, the peculiar quality of the psychical experience which we mark off as active, strongly suggests that muscular sensations are not wholly correlatives of afferent nerve-processes. If every psychical difference of quality is to have its corresponding physiological difference, there is some *a priori* reason for correlating the great and profound difference between passive sensation and active consciousness with the most important and radical difference observable in the nervous process—viz., that between the afferent and the efferent current. As, however, the subject of muscular sensation has been discussed so recently by this Society I should be sorry to complicate this evening's topic by raising again this difficult issue.

I will pass, then, to the question, whether what we all know as the mental attitude of attention appears subjectively identical with muscular consciousness.

Now, I think that if we carefully examine our state of mind when we are attending to impressions or ideas, we are in some cases able to distinguish attention as something apart from and independent of muscular activity. Thus, as everybody knows, we may fix the eye on an object and yet not attend to that object. Helmholtz notices the fact that we can attend to an object in the side regions of the field of vision without fixating the object. Where, one may ask, is the motor factor, the process of muscular adjustment in this case?

It may no doubt be said that there is a *tendency* in this case to move the eye in the direction of the object. But this tendency may, I believe, be completely counteracted without any injury to the process of attention itself.

In this connection I should like to refer to a personal experience that befel me while I was thinking over this

subject. I was walking along a narrow lane in a fog, lost in thought. I came on a lamp which shot its rays through the fog. I involuntarily stood still and fixated the lamp, thinking all the while intently on my psychological problem. When this intellectual effort relaxed, and not till then, I saw and recognised the lamp. Now here was energetic muscular action, and equally energetic concentration; but what was the relation between the two? That this involuntary assumption of the attitude of the seer, of the fixed head, convergent eyes, &c., somehow aided the process of mental concentration is certain. But was this muscular adjustment the *whole* of the process? If so, I ought surely to have been mentally occupied with no abstruse problem of psychology, but with the concrete sensible object before my eyes, even though it had been far less brilliant and imposing an object than the lamp. It struck me that I had, by pure good fortune, lighted on a case where the two phenomena, though both present, were rendered exceptionally distinct. The whole pose of body, head, &c., was for seeing; yet I saw not because that which seems something else, and to which I give the separate name of attention, was elsewhere.

The same partial independence of the process of attention and of the motor process appears, too, in ideational attention. If I think, for example, of a circle, with the eyes closed, I am aware of what Dr. Bain calls a control of the muscular element. I not only have muscular sensations which tell me that the peripheral organ by means of which I acquired the idea is engaged, but I am aware of a motor impulse to retrace the curve of the circle. It has been observed by N. Lange that in recalling the image of an object there are commonly observable movements of the eye corresponding to the contour of the object.[1] But now let us suppose that I am trying to visualise not any particular form, but merely some shade of colour—say peacock-blue. In this case, I certainly find the motor element much less prominent. I am hardly aware, indeed, in this instance, of any sensation of ocular strain, and should rather say that the eyes were in the easy and natural possession, described by Helmholtz as

[1] Quoted by Wundt, *Physiol. Psychol.*, ii. p. 242.

the primary position. Nor does attention in this case resolve itself into a renewal of the muscular action concerned in uttering the name of the colour. Indeed, I find that any thought of the name in this case, so far from helping, distinctly disturbs the visualising process. I must for the moment be all internal vision. And yet, though the muscular element in this case is, to say the least of it, considerably reduced, the active consciousness, the attention, is as clearly present as before.

On purely subjective grounds, therefore, I should be disposed to say that attention, though always accompanied by a motor process, which last, through the concomitant muscular sensations, undoubtedly lends something of its characteristic colouring to the mental state, cannot wholly be resolved into a motor phenomenon. The relation of the two is, I suspect, very similar to that which obtains between an emotion and the several sensory and motor phenomena which accompany it. Fear is always accompanied by characteristic physiological changes, involving the organs of circulation, respiration, and the muscles; and fear would not be fear but for these processes which contribute, in the sensations to which they give rise, characteristic features to the mental state. Yet when Professor W. James, of Harvard College, not long since proposed to prove that emotion is nothing but the result of the organic changes and correlated sensations, most persons probably regarded the proposal as paradoxical.

(2) Is this conclusion, reached by subjective analysis, confirmed by a consideration of the known effects of attention? And here we may confine ourselves to the most immediate and palpable effect of attention—the intensification and definition of the sensation or idea.

In the case of actual sensations obtained through the special sense-organs, the motor process must of course be pronounced sufficient. That the muscular adjustment of the eye and head for direct and clear vision, of the ear and head for sounds of a particular direction and definite pitch should help to strengthen and define the sensation, is just what we should expect. Yet even here muscular adjust-

ment does not in all cases—for example, that of attending to an object in the side region of the field of vision—seem to be an adequate cause of the phenomenon.

Again, in the case of attending to organic sensations, it is easy to see that a motor process may be present, which would serve, to some extent at least, to produce the observed effect. If, as is generally allowed, we learn to localise our bodily sensations by help of movements, which bring the hand to bear directly or indirectly on the particular region of the organism, it would seem to follow that in fixing consciousness on any part, motor elements of a nascent character may concur.[1] Yet it hardly seems likely that such a faint motor constituent could effect the remarkable results here observable. And, indeed, as I understand, physiologists refer the phenomena mainly, if not exclusively, to another process, also motor, though not implicating the so-called voluntary muscles, by which the circulation of the part attended to is modified.

Let us now come to the more difficult case of ideas. The theory that a motor process effects the fixation and increase in vividness and clearness of the idea seems to imply that in attending to an idea there is a process of motor innervation corresponding to that which subserves the development of the correlative sensation, and involving therefore the particular peripheral organ concerned. Thus, in seeking to visualise a colour the ocular muscles are supposed to be innervated. The further explanation of the effect as given by Wundt and others is as follows. As the result of the innervation of the ocular muscles, muscular sensations arise, which, being associated with the original sensation of colour, serve to reinstate this in a much more vivid ideal form.

In justice to the view that would resolve the whole of attention into a motor process, I may point out that the effect of motor innervation on ideation may be wider than is here suggested. If, as is fairly certain, the ideational

[1] It is worth noting, perhaps, that the range of co-operation of moor elements is materially increased by the supposition that there is an innervational stage of active or motor consciousness, for this might arise by means of a weak motor discharge which does not involve actual muscular contraction.

centre stands in the closest organic connection with the corresponding sensational—if indeed the two are to be regarded as distinct—and this last again, in the closest organic connection with the motor centre for the sense-organ, it seems conceivable and probable that any excitation of this motor centre, even if it did not effect actual contraction of muscle and reflex muscular sensation, would react on and reinforce the activity of the ideational centre.[1]

It may, I think, be granted that we never think of a colour, sound, or smell without some amount of innervation of the muscles of the corresponding peripheral organ. Subjective reflection tells us that this is so. But as we have seen, the motor process involved seems, in certain cases, to be a very faint one. It is to be observed, too, that the muscular sensations most characteristic of ideational attention or thought are not those of the sense-organ concerned, but of the head. According to Fechner they are the concomitants of contractions of the muscles of the skin on the occipital region of the head.

It has recently been suggested by Münsterberg, in the work already alluded to, that these muscular sensations of the head are concomitants of that fixation of the head which is so important a factor in the muscular adjustment involved in looking or listening. But so far as I can observe, the actual operation of looking or listening is not accompanied by any corresponding contractions of the muscles of the skin of the head.

Let us now inquire more particularly into the effects of such muscular action in the peripheral organ as is known to take place in ideational attention. That the innervation of the ocular muscles does favour visualisation is I think proved by a number of facts, more particularly that steadily gazing at the fire, the distant horizon and so forth, tends to develop a state of vivid reverie, which in extreme cases may rise to the strength of a hallucination. But I think it is easy to see that the whole effect in these cases is not due to the muscular sensations.

[1] Since writing this I have noticed a passage in Dr. Ferrier's *Functions of the Brain* (second edition, p. 462), which seems to me to recognise this direct action of motor on sensory centre.

And here it is important to distinguish between those sensational and corresponding ideational elements which merely involve a *general* muscular adjustment of the sense-organ concerned, and those which in addition involve a *special* muscular action. Thus in the case of visual elements we have colours which simply presuppose adjustment of the eye for seeing under particular conditions, and forms which involve definite muscular movements of the organ corresponding to the lines composing the form. That in recalling the form of a circle, the renewal of the muscular action should tend to reinstate the idea is just what might be expected. It may be remembered here that J. S. Mill made use of this circumstance in explaining our intuitive certainty with respect to the truth of geometrical axioms. We can at any moment, by ocular movement, re-create the experiences out of which our first ideas of straight lines, circles, and so forth were extracted.

In the other case, represented by the visualisation of colour, the adequacy of the muscular factor seems very doubtful. It has been ascertained by Mr. F. Galton, that many persons can visualise colours quite as vividly and distinctly as forms; and though no doubt individuals differ greatly here, some at least have the power of rendering colour images vivid by a prolonged effort of attention. Yet in this case there is no muscular element corresponding to that present in tracing out the contour of a particular form. That innervating the ocular muscles should react on the activity of the visualising sensory centre is, as I have allowed, quite understandable. And if this centre is at the moment in a state of sur-excitation corresponding to the idea of a particular colour, such innervation would no doubt do something to increase the excitation. But I should find it hard to believe that it could produce that very considerable augmentation of the activity of the centre, *in one direction only*, which appears to be involved in the process of reinvigorating a faint visual image by an effort of attention.

It seems to me, then, that there is room for a further supplementary hypothesis with respect to the nervous process involved in attention. Assuming what Dr. Ferrier and others

appear to be agreed upon, that attention specially involves the frontal lobes, which contain some at least of the centres for movements of the head and eyes, the question arises whether this particular region may not stand in a closer, more penetrating and more complex connection with all those regions of the brain which constitute the centres of sensation and ideation, than is involved on the theory of its being merely the centre for particular groups of movements. Wundt, it may be remembered, while fully recognising the part played by motor processes in the intensification and fixation of sensations and ideas, postulates a further nervous process, viz., a centrifugal impulse from the centre of attention or apperception to the sensorium, which is simultaneous and organically conjoined with another centrifugal process, viz., that of motor innervation issuing from the same region.

How such a process would have to be conceived it is for the physiologist to say; whether this action of the highest regulative centre on sensory centres should include an inhibitory as well as a stimulatory effect; whether it is wholly direct, or whether in part indirect, involving the control of the circulation in the cerebral capillaries through the vaso-motor system—these are points on which it would ill become anybody but a trained neurologist to offer conjecture.

Biological Conception of Attention.

In bringing to a close a paper which I fear may strike you as a little discursive, and already long enough, I should like to add a few words on the biological function and the practical aspect of Attention.

In the first place, then, it is evident that attention, by limiting and selecting, substitutes in our mental life a relative simplicity in place of a confusing multiplicity of elements. If, under the crowd of sensory stimuli by which we are assaulted, we had no power of selective fixation, consciousness would be nothing but a confused mass of sentience and adjustment of organism to environment would be impossible. In the highly complex life of man, as distinguished from that of the lower animals, and more particularly of civilised man,

such a power of selective reinforcement of particular elements presented, corresponding to the predominant interest of the moment, seems to be indispensable. The power of mental concentration is thus biologically correlated with the multiplex action of the environment on the human organism, and the multiplex form of reaction necessitated by this.

In the second place, all attention is in its nature temporary and self-terminating, and this not merely because we cannot indefinitely maintain the effort of attention, but because by attending now we render attention by-and-bye unnecessary. In other words, the severer exertions of mind involving exceptional expenditure of nervous energy are required only in the earlier and more difficult stages of adjustment. That this arrangement implies an economising of force is obvious.

One can understand, too, from this what is often spoken of as the special achievement of genius—the rapid mastery of a new acquisition or group of acquisitions by a supreme effort of concentration. Great mental power, which a number of writers connect with exceptional concentration, may perhaps be said to imply exceptional capacity for new adjustment through initial concentration of the most intense and vigorous kind. Such exceptional power of concentration has for its probable condition a special development of particular nervous centres, by which they become capable of frequent fits of strenuous activity.

The effect of such exceptional power of attention is to expedite the processes of intellectual assimilation, and thus to open the way for new lines of adjustive activity. Hence, as we see in the case of the greatest minds, as Goethe, the enormous range of intellectual adjustment possible. Such men, because of the splendid stock of nervous energy with which they are endowed, and also of the remarkable results of successive acts of concentration, lead the intensest lives, and are conscious in a sense compared with which the narrow stationary man of routine may be called unconscious.

Thus, the few fortunate master minds, so long as they preserve a sound digestion and other sadly-despised bodily conditions. For most men, however, what stock of con-

centrative power nature has conferred on them is apt to be limited in its direction. As was pointed out in the earlier part of this paper, attention follows the lines of interest, and for most of us interest is apt to narrow itself down, as life advances, to a few circumscribed regions. The specialization of professional work, so characteristic of the social condition of our age, compels attention itself to become the slave of that very Habit of which it is the natural enemy. The mind has to retrace again and again the same path, face again and again the same harrassing problems. And here I trust I may not seem intruding on the proper domain of the pathologist if I remind you that in this very circumstance lies one of the chief dangers to mental health. Under the pressure of fixed interests the power of attention which was developed by school discipline, and the ample indulgence of which was the chief delight of youth and early manhood, loses its alacrity, its freedom of movement. The man no longer cares to master a new subject. He listens, he reads, only as opportunity occurs to enrich his own narrow domain of ideas.

Wise men recognise the danger, and save themselves from the tyranny of paramount interest by keeping the range of interest as wide as possible. A busy professional man who can make the effort of tearing himself for the moment from daily absorbing topics and throw himself into politics, art, music, or even the lightest vein of fiction, has a safeguard against worry and other more serious evils. In view of the importance of retaining unimpaired the power of a wide impartial attention, it were well, perhaps, to direct the work of education more expressly than is done to the problem of developing this power. A boy might with advantage be exercised in concentrating on a few special subjects and at the same time in acquiring a certain knowledge of others. We seem in education, as in other things, to be drifting into extreme specialisation. The reasons are obvious. It is only by such narrow restriction of the mental gaze that exact detailed knowledge becomes possible. But it may be as well to recall what J. S. Mill said in his St. Andrew's Address, that an educated man should combine a minute

knowledge of one or a few things with a general knowledge of many things. What Mill and others have urged in the interests of a liberal culture may also be urged in the interests of mental health. To be able to take wide excursions in the field of knowledge far beyond the boundary of our own small plot is at once a sign and condition of the *mens sana:* a sign, because it shows there is as yet no morbid preoccupation with one subject; a condition since it is by continuing to exercise this power that we shall best arm ourselves against the insinuating attacks of such preoccupation.

ON ASSOCIATED EYE-MOVEMENTS PRODUCED BY CORTICAL FARADIZATION OF THE MONKEY'S BRAIN.

WITH A DIAGRAM.

BY F. W. MOTT, M.D., AND E. A. SCHAEFER, F.R.S.

(*From the Physiological Laboratory, University College, London.*)

OUR experiments upon the associated eye-movements which result from cortical faradization may be described under four heads, viz. :—

1. Associated movements produced by unilateral faradization of the frontal region of the cortex.

2. Effects of bilateral faradization of the frontal cortex.

3. Effects of bilateral faradization of the cortex of the occipital lobes.

4. Effects of simultaneous excitation of the occipital lobe of the one, and of the frontal lobe of the other hemisphere.

I.—*On the associated eye-movements which are caused by unilateral excitation (weak faradization) of the head and eye area of the cortex of the frontal lobe.*

Since these movements were first investigated in the monkey by Ferrier,[1] very little advance has been made in our knowledge of the subject. Ferrier, as is well-known, obtained, on placing his electrodes over a certain area of the frontal lobe which he has marked (12) conjugate deviation of the eyes with simultaneous movement of the head to the opposite side. Horsley and Schaefer[2] showed that this area must be extended to the marginal convolution above, and nearly to the lower end of the pre-central sulcus below, but otherwise obtained no constant differentiation of the eye-movements (*e.g.*, upwards or downwards as well as sideways) from excitation of the different parts of the area,

[1] *Proc. Roy. Soc.*, 1875. [2] *Phil. Trans.*, 1888. B.

(although such differentiation is obtained on excitation of different parts of the occipital visual area.[1] Horsley and Beevor[2] subsequently made a minute investigation of the area as thus defined. They showed that the strongest lateral movements of the eyes are got by excitation in the angle of the pre-central sulcus[3] but otherwise for the most part failed to obtain a differentiation of the movements. Such upward inclination as they noticed was "very rarely observed," and a downward movement "was noticed but twice" in a very large number of experiments. Moreover, the points to which they localize these inclination movements do not at all correspond with the results of our experiments. Whether this difference of result is due to our having employed monkeys belonging to other genera, which is hardly likely to be the case, or to sources of error which we shall presently have to indicate, we need not, in consideration of the rarity of the results they record, here discuss.

Very early in the course of our investigations we were driven to the conclusion that it was useless to attempt a differentiation of the eye-movements unless monkeys with exceptionally large and well-developed brains were employed. These were difficult to procure, but ultimately we were successful in obtaining a number of large specimens of Callithrix, in which we were able to obtain the desired differentiation with comparative precision. Very definite confirmatory results were also got in one well-developed Bonnet monkey, and in a large Rhoesus; whereas in smaller Rhoesus and Liponda monkeys either no indications of upward and downward movements could be got, or such as were obtained were indefinite or contradictory at different periods of the experiments. This is probably to be ascribed to the fact that, at different periods of an experiment, the different parts of the head and eye region are unequally excitable, either in consequence of difference in etherization, or of

[1] Schaefer. BRAIN, April, 1888.

[2] *Phil. Trans.*, 1888. B.

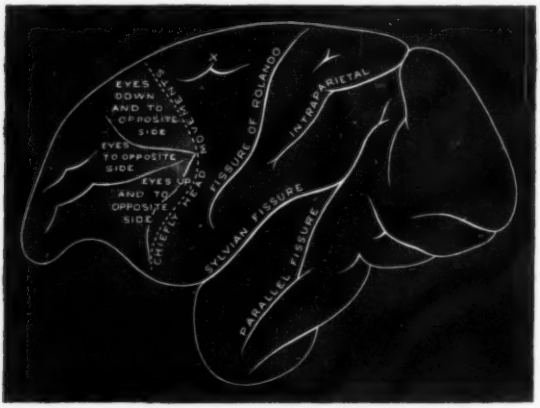
[3] This part of the eye area they have termed the centre of "primary representation" of the simultaneous movements of the head and eyes. We must, however, confess that we have been unable to comprehend the distinction implied by this term.

vascular disturbance consequent on exposure; and when the effect of the excitation has but a small area to spread over, it may tend to call into play those parts of the area which are at the moment more excitable. It is well-known to all who have investigated the "motor" cortex by faradization that anomalous results are frequently obtained in consequence of such increased excitability of a particular area (or of its connections in the lower or bulbo-spinal centres), *e.g.*, after a particular point has been faradized for a short time and thus thrown into a condition of increased excitability, it often happens that faradization of a neighbouring area will produce not only the effect rightly due to stimulation of that area itself, but also those which had been previously obtained from the area which had before been faradized. In this manner, if from any cause, the part of the head and eye area which is concerned with the production of upward movement of the visual axes has been rendered more excitable than that concerned with downward movement, minimal excitation of almost any part of the area, will produce upward combined with lateral movement; or *mutatis mutandis* downward, combined with lateral movement, especially if the brain be small, or the area less expanded than usual. This is one important source of error which must always be taken into consideration. Another one is to be found in the fact that, if the eyes are in the first position at the moment of excitation, and the faradization of the pia mater produces awakening from narcosis; there is then almost invariably a tendency to raise the eyes and elevate the upper eyelids, whatever be the part of the cortex stimulated. Movements of the eyelids sometimes produce the appearance of upward or downward movement of the globe when there may be only a purely lateral movement, and this is a source of error which also requires to be guarded against.

Lastly, in all experiments having for their object the determination of any upward or downward inclination combined with the conjugate lateral deviation of the visual axes, it is essential that the eyes at the moment of faradization should be in the primary position; for if they are already deflected downwards or upwards, the excitation of a spot

which would bring them from the primary position into the simple lateral secondary position, will bring them into the same (simple lateral secondary) position, and will consequently produce an upward or downward deflection combined with the lateral movement.

Some of these sources of error also complicate the investigation of the occipital (or sensory) visual area of the cortex; but this area is so greatly expanded that the difficulties are much diminished, and hence the determination of the differentiation of different movements has been far easier than in the less expanded frontal region.



The general results we have obtained from unilateral faradization of the head and eye area in Callithrix were demonstrated to the Ophthalmological Society on March 26th. They show that as regards conjugate deviation of the eyes this area may be regarded as consisting of three zones (see diagram), viz., (1) a middle zone immediately below the horizontal part of the pre-central sulcus, faradization of which is followed by simple lateral deviation, well-marked and without either upward or downward inclination; (2) an upper zone immediately above this, which may extend to and include part of the marginal gyrus, giving on

faradization downward inclination, usually combined with lateral deviation; and (3) a lower zone immediately below the middle one, and sometimes extending nearly to the lower margin of the hemisphere, which gives upward inclination, usually also combined with lateral deviation. When all three effects are well differentiated the zone which gives a purely lateral deviation is comparatively narrow; rarely, however, so narrow as that on the mesial surface of the occipital lobe, where zig-zagging lateral deviation is sometimes obtained, showing that it is little but a line of demarcation between upper and lower zones.[1] Anteriorly the middle zone tends to broaden—indeed all the zones become broader and the effects more indefinite, as the electrodes are carried forwards into the pre-frontal region.

We have not carried out observations upon the head-movements so systematically as upon the eye-movements, because these unilateral movements lay somewhat beyond the immediate scope of our enquiry, which was concerned with bilateral movements only. We have, however, observed a very definite rotation of the head to the opposite side and downwards, accompanying the downward inclination produced by excitation of the upper zone, and a rotation upwards and to the opposite side, accompanying the upward inclination produced by excitation of the lower zone, besides the simple lateral movement of the head which usually accompanies simple lateral deviation of the eyes. These head movements are most marked as the posterior limit of the head and eye area is approached; and at this limit they are frequently accompanied by movement of the arm at the shoulder, or by contraction of the muscles of the face and head. A movement of the opposite ear (pricking) is almost constantly a concomitant of the lateral deviation of head and eyes which follows excitation in the angle of the pre-central sulcus.[2]

Lastly, we have investigated the effects of unilateral excitation of different parts of the area after section of the corpus callosum, and after destruction of the corresponding

[1] We have, however, once obtained a zig-zagging lateral deviation on excitation of the middle zone of the frontal area.

[2] *Cf.* Horsley & Schaefer. *Loc. cit.*, p. 8.

area in the opposite hemisphere. Our results here have only served to confirm those of previous observers in shewing that the bilateral movements continue after as before the lesion, and that the associated effect is not necessarily produced through the corpus callosum and the cortical centres of the opposite side of the brain, but possibly through lower centres.

II. *Effects of Bilateral Faradization of the Frontal Cortex.*

So far as we are aware, no previous observers have investigated the effects of calling into activity identical "motor" cortical centres of both hemispheres by simultaneously faradic excitation. For the ordinary limb-centres, which produce only unilateral movements, such investigation has no particular interest, but for the conjugate movements of the eyes the case is different and it would be difficult to predicate the result which is to be expected.

The method which we at first adopted was to pass the same faradic current through two pairs of electrodes, which were applied to similar points on the two frontal lobes. In this manner a stimulus of as nearly as possible the same strength is applied on the two sides, and it might be supposed that the effect produced would be equal. This is, however, rarely the case; almost invariably the action of one side preponderates over that of the other. This is due to the fact that the excitability of the two sides is scarcely ever the same, so that a minimal stimulus which would excite one side would produce no appreciable effect on the other. In order to overcome this difficulty we had to adopt a different method, viz., to employ two completely distinct induction coils and electrodes, the keys being so arranged that either one or other side of the brain could be separately stimulated, or both could be excited simultaneously. Points were in the first place found on the two sides which gave when separately stimulated exactly the same kind of conjugate deviation of the eyes (but of course in different directions), and by shifting the secondary coils, the extent and rapidity of this movement was also got as nearly as possible equal—so far as could be judged by the eye. These preliminary deter-

minations having been made, two points were, after a brief period of rest, simultaneously excited.

When two points—which give on unilateral stimulation simple lateral conjugate deviation of the eyes—are thus simultaneously bilaterally stimulated, the result is almost invariably, if the eyes are not already in the primary position, to bring them into this position and to produce fixation of the visual axes upon a distant object. Sometimes there is a slight convergence of the visual axes, and invariably, if they are previously divergent, as is often the case in narcosis, this divergence becomes obliterated and they become fixed in a parallel or slightly convergent direction. If at the moment of simultaneous bilateral excitation the eyes were exactly in the primary position, they almost invariably remain motionless. The main result therefore of equal simultaneous bilateral excitation of points, which when unilaterally excited cause simple conjugate lateral deviation, is to produce visual fixation, without any tendency to lateral deviation. If the two pairs of electrodes are placed on the upper or lower zones of the frontal area, that is to say, on similar points, which when unilaterally stimulated cause conjugate lateral deviation, combined respectively with more or less downward or upward inclination, the effect of simultaneous bilateral excitation is to cause a simple downward or upward inclination of the visual axes, as the case may be, usually with maintenance of parallelism of the axes, sometimes with slight convergence followed by fixation.

Sometimes there is a struggle between the two centres, the eyes tending first slightly in one direction and then in the other; finally, in these cases they usually tend eventually to deviate to one side. This result is, however, much more rare than those above described. Whenever an immediate lateral deviation to one side was obtained, we assumed that the bilateral excitation was unequal, and generally found that by diminishing the strength of the current which was applied to the opposite cortical centre, we could obtain the usual result. One of the most remarkable results of bilateral faradization is the modification which is frequently produced in the results of subsequent unilateral excitation.

In very many instances we have found that after even a short period of bilateral faradization, which has produced the parallelism or slight convergence and fixation of the visual axes in the manner above described, unilateral excitation does not produce the usual effect of conjugate deviation of the eyes to the opposite side, but is followed by exactly the same result as the preceding bilateral excitation, that is to say, it produces or continues the condition of visual fixation. It is, in fact, as if the lower centres had been set by the bilateral excitation in a particular groove or habit of action, from which they do not immediately return to the indifferent condition.

III. *Effects of Bilateral Faradization of the Occipital Visual Area.*

The results obtained by us from bilateral excitation of the occipital lobes, are in all respects comparable to those yielded by stimulation of the frontal areas. Equal and simultaneous excitation of points, which give when unilaterally stimulated a simple lateral conjugate deviation, produces parallelism and fixation of the visual axes, and if the eyes are already in the primary position the stimulation is usually unaccompanied by any movement. If, however, the electrodes are placed on those zones which give, on unilateral stimulation, downward or upward inclination of the visual axes combined with lateral movement, the fixation on bilateral stimulation is then preceded by downward or upward inclination, without any lateral deviation. There may also be produced, as with the frontal area, slight convergence of the optic axes.[1]

IV.—*Effect of Simultaneous Excitation of the Occipital Visual Area of the one Hemisphere and of the Frontal Area of the other Hemisphere.*

If the electrodes be placed, the one on a point of one frontal area which gives simple lateral deviation of both eyes to the opposite side, the other on a point of the opposite occipital lobe which gives the same movement of the eyes

[1] Schaefer, "Experiments on Electrical Excitation of the Visual Area," BRAIN, April, 1888. Also Obregia, *Ueber Augenbewegungen auf Sehsphaerenreizung,' Arch. f. Anat. u. Physiol., Physiol. Abth.*, 1890.

but in a contrary direction, the strength of the stimulus being so graduated as to produce in each case about the same amount of deviation when separately stimulated, then when these two points are simultaneously excited, the action of the frontal cortex invariably preponderates, so that a simple effect of lateral deviation is obtained, the same as would be produced by excitation of that frontal centre alone. To balance this preponderating action of the frontal centre it is necessary enormously to increase the strength of the stimulus which is applied to the occipital lobe. This result is in conformity with the difference of the period of latent stimulation for the frontal and occipital lobes respectively.[1]

[1] Schaefer, *International Monthly Journal of Anatomy and Physiology*, 1888.

ON MOVEMENTS RESULTING FROM FARADIC EXCITATION OF THE CORPUS CALLOSUM IN MONKEYS.

BY F. W. MOTT, M.D. AND E. A. SCHAEFER, F.R.S.

(*From the Physiological Laboratory, University College, London.*)

If a pair of fine electrodes, guarded except at the extremity with shellac or paraffin, are carefully passed vertically into the great longitudinal fissure between the hemispheres and are thus used to stimulate the upper surface of the corpus callosum, bilateral movements of the head, trunk and limbs are obtained according to the point to which the electrodes are applied. These movements are due to excitation of fibres in the corpus callosum and not to a spreading of the electric currents to the "motor" surface of the cortex, for (1) the effects are obtained with very weak currents, and (2) if the electrodes are somewhat withdrawn from the surface of the corpus callosum the effects immediately cease, although the excitation is now applied at a point nearer the motor surface.

The effects are obtained, roughly speaking, in the following order from before back, viz.:—When the electrodes are applied over and just behind the genu, head and eye movements are produced; further back, movements of the arms at the shoulder and of the upper part of the trunk; then movements of the forearms and general movements of the hands and fingers; then of the lower part of the trunk and tail, and lastly, movements of the lower limbs. The movements are not obtained if the anæthesia be too deep; they become uniiateral if the grey "motor" cortex of one hemisphere be destroyed by actual cautery, and if the excitation be too strong or too prolonged they pass into epileptoid contractions; they are evidently due therefore to indirect excitation of the

"motor" cortical centres. Although movements of particular parts of the body are thus roughly or generally capable of being produced by excitation of different parts of the corpus callosum the localization is not exact, for there are usually other movements produced at the same time but less markedly, as if the fibres going to the several cortical areas, although more massed at certain parts of the corpus callosum, are yet scattered over a considerable length of the commissure. This is in conformity with the degeneration results obtained in the corpus callosum by Sherrington, after unilateral destruction of localized portions of the "motor" area.[1]

In order to investigate more precisely the position of these fibres in the corpus callosum we have in several experiments cut away one hemisphere and directly excited the cut surface of the corpus callosum. The movements then obtained are of course confined to the side of the body which is in nervous connection with the intact hemisphere, that is to say, if the left hemisphere be cut away, the movements are on the left side of the body.

Operating in this manner we have been able to obtain much more distinct and specialized movements than when the intact commissure is stimulated at the bottom of the longitudinal fissure, but the experiment is far more difficult to perform in this way, and is frequently entirely vitiated by the profuse hæmorrhage which usually follows the removal. The general results are, however, the same. Movements of the eyes and head are got most anteriorly, of the lower limb most posteriorly; and of the upper limb and trunk between. With the weakest possible excitation, a favourable slight condition of narcosis, and a freshly-made section of the commissure we have occasionally obtained very precisely specialized movements, such as a simple conjugate lateral deviation of the eyes, simple opposition of the hallux and the like. These simple movements were, however, rarely got; usually although one part showed a greater tendency to move, other and even distant parts of the body might also but less strongly be set in action, thus indicating as before, a scattering of the commissural fibres. Even when the movements are confined

[1] *Journal of Physiology*, vol. x.

to one part of the body or one limb, they are very seldom completely specialized, but are usually general movements. This is the case, not only when the cut surface is stimulated, but as might be supposed also when the excitation is applied from above to the intact corpus callosum. For instance, the flexors and extensors of a limb are often both thrown simultaneously into action, and the eye-movements instead of being definite movements of conjugate deviation are frequently indefinite or rolling movements. Nor, in the case of the stimulation of the intact corpus callosum, are the muscles which are called into action always quite the same on the two sides of the body, although those which are most strongly affected always belong to the same part. This may easily be understood, if we consider that the bundles of fibres which pass within the corpus callosum to corresponding points in the grey cortex of the hemispheres would not necessarily cross at exactly the same point, for those passing to the one side might be placed more superficially or more anteriorly as compared with those passing to the other side: hence we should expect to find some differences in the movements of the two sides.

For some reason which we are unable to explain, we were never successful in obtaining contractions of the muscles of the face from stimulation of any part of the commissure.[1]

It is not improbable, however, that the commissural fibres for their motor centres (and perhaps also for those of the larynx) may yet be found in the rostrum of the corpus callosum. We certainly explored this part on two or three occasions, but the negative results we obtained may perhaps be ascribed to the hæmorrhage which accompanied its exposure. This may also account for the fact that we failed to get any movements on stimulating the splenium, although we had expected to obtain movements of the eyes from its excitation. Practically all the results which we have obtained have been yielded by the thinner middle part or body of the great commissure. We have performed a con-

[1] We were also unsuccessful in obtaining any independent movements of the fingers, although we once or twice obtained such movements of the toes.

siderable number of experiments and have employed for the purpose several different kinds of monkey (Liponda, Rhoesus, Bonnet, Callithrix). The most precise evidence of localization within the commissure which we obtained in any was got from a fine, intelligent Bonnet; but the conditions of experiment may have been accidentally favourable on this occasion. The sequence of movements obtained by stimulating the cut surface from before back was very distinct in this case and certain movements were easily isolable. In most other animals the movements were, as has been above mentioned, of mixed character, with occasional preponderance of movement of particular parts. Thus in one case retraction of the arm and shoulder, combined with flexion of the leg and hip, recurred as a prevalent action. In another case, in which we stimulated with flat paraffin-guarded electrodes successive points of the under surface of the corpus callosum, we obtained along the whole length of the excitable part, movements of the eyes and eyelids.

The general results of our experiments and the inferences which may be drawn from them are briefly the following:—

1. The corpus callosum contains fibres excitation of which produces movements on both sides of the body.

2. These fibres do not pass directly from the corpus callosum to the internal capsule[1] but enter the grey cortex of the hemispheres, and there come into connection with the so-called "motor centres."

The fibres which pass to the several motor centres, although mainly massed in definite parts of the corpus callosum, are not confined entirely to those parts, but are to a certain extent scattered along the middle thinner part of the commissure.

[1] When destruction of the grey cortex was effected by actual cautery, as before described, care was taken to avoid injuring any connection between the corpus callosum and the internal capsule.

A CONTRIBUTION TO THE STUDY OF CHEYNE-STOKES BREATHING.

BY J. DIXON MANN, M.D., F.R.C.P.,

Physician to the Salford Royal Hospital. President of the Manchester Pathological Society.

THE phenomena comprised under the designation Cheyne-Stokes breathing, in a comparatively short time have called forth a copious literature on the subject, derived both from experimental and from clinical observations. Many hypotheses have been advanced explanatory of the periodic alterations in breathing, but as yet without general acceptance being accorded to any. I have recently had under continuous observation a case, probably unique, in which Cheyne-Stokes breathing lasted for more than a year. It afforded an admirable opportunity of studying the symptoms during life, and of searching after death for pathological changes affecting the organs concerned in their production. Since the subject is involved in obscurity, it is possible that symptoms which at present appear to have no relation to the phenomenon in question, may in the future aid in its interpretation, I therefore present, in as few words as possible, a full report of the case.

The patient at the time of his death was seventy years of age. He never enjoyed robust health, but, until the latter period of his life, he never had any illness of importance. There is nothing in the family history that has any bearing on the case. The patient was very careful and abstemious in his habits. He was endowed with the highest type of mental capacity, and until the age of fifty he had exercised his mental powers to the fullest extent in original scientific investigations, necessitating much profound thought and elaborate calculations. My first professional relation with

him was in 1874. Previous to this time, and for several years after, he suffered from attacks of profuse epistaxis. For two years his general health had gradually deteriorated without any decided symptoms, and he became depressed in spirits, displaying much anxiety about his condition; afterwards he became more cheerful. About this time, the patient used to complain of an uncomfortable sensation at the base of the brain, which he localised by opening his mouth and pointing with his finger to the posterior part of the palate, explaining that the sensation was inside the head. Still later he suffered from a feeling of confusion in the head, with some amount of pain in the parietal regions, which, however, was not violent.

In 1880, he developed a soft mitral systolic murmur, which could not be heard beyond the anterior axillary fold. The left ventricle was subsequently somewhat hypertrophied, but the apex beat never descended more than half an inch below the normal position. The enlargement was much less than might have been expected from the atheromatous condition of the arteries found after death. The heart beats were always very regular; the pulse never partook of the character of the mitral pulse, neither in smallness nor in intermission. As will be mentioned later on, there was an occasional intermission, but it was accidental rather than habitual. The condition of the heart never affected the breathing, the regurgitation evidently being slight. The lungs were healthy, the breathing powers being excellent. The patient never suffered from any bronchial affection.

In 1887 he had an attack of partial paralysis of the right arm and leg, with transient motor aphasia. In a few weeks the paralysis almost disappeared. The aphasia passed off in a few days, but the speech remained less distinct than before. The mental powers had been gradually diminishing, and their downward progress was hastened after this attack. The epistaxis had ceased to recur for some time before the paralysis, and never returned. On August the 5th, 1888, after a considerable amount of cerebral excitement, which came on without external cause, he entirely lost the use of the right side. The hemiplegia was complete, and included

the lower part of the face on the right side. The tongue on protrusion was pushed over to the paralysed side, the right angle of the mouth dropped somewhat, and the masseter of the same side was flabby. The upper facial muscles were unaffected. He could without difficulty eat food which had been prepared by being finely divided, but he had little power of mastication. So far as could be made out, the organs of special sense were not affected. This attack was accompanied by complete motor aphasia, which was permanent, the patient never regained the power of speech. On account of the aphasia and the failure in the mental powers, no reliable evidence could be obtained as to sensory disturbance. In a few hours after the attack, some amount of motility returned in the leg, the arm remaining useless.

On August 19th, 1888, he began to breathe irregularly, and, during my absence from home, he was seen by my colleague, Dr. Edge, who found he had well-marked Cheyne-Stokes breathing. On my return, two days after, his condition was much as before, with the addition of the Cheyne-Stokes breathing, which continued. In a few weeks the general condition improved, and he became sufficiently strong as to walk unaided; the arm also improved, but never recovered much power of movement. The condition at this period and for some time afterwards was as follows:—Pulse from 64 to 72, regular and not excessive in tension; appetite excellent; bowels obstinate, requiring constant attention; sleeps well; skin dry and cold, there was never excessive perspiration. Sensation appeared unaffected throughout. There was no special atrophy, although the muscles generally were wasted. There was no rigidity. The knee-jerk was slightly increased for a time on the right side, afterwards was abolished on both sides. The skin reflexes were early lost on the right side. The left leg was often moved about for hours together when the patient was in bed, apparently as an outlet for cerebral irritability. The urine, usually amounting to 40 to 60 ounces in the twenty-four hours, was of medium colour, free from deposit, s.g. 1,010 to 1,016, no albumen, urea from 350 to 385 grains in the twenty-four hours. On several occasions a considerable excess of chromogen was present in

the urine, probably indol, with some other substance, the oxidised product of which was not soluble in ether. When the urine was treated with hydrochloric acid and bleaching powder it yielded a claret-colour, and on being shaken out with ether, imparted a blue which gave the absorption spectrum of indigo-blue. No relation could be traced between this condition of the urine and any symptoms present at the time. There was never any dropsy, nor even œdema of the ankles. The bladder was unaffected. The patient could protrude the tongue to the full extent, could masticate feebly, and could swallow both solid and liquid food without difficulty. There was never any tremor of the tongue, nor of the lips, nor hands. At one time bulbar symptoms seemed imminent, but did not advance beyond some difficulty in swallowing, necessitating the food being introduced to the back of the mouth. The bulbar symptoms disappeared in about a week. The only vocal sounds the patient could produce were "ai" and "o," meaning yes and no. He could not read, but liked to look at the pictorial papers. He had occasional attacks of sickness, which resembled "bilious attacks" more than gastric crises. During all this time the mental condition progressively deteriorated, paroxysms of restlessness and irritability occasionally manifesting themselves. With slight fluctuations, the physical condition underwent no perceptible change for several months. The patient was dressed each day, and with a little assistance walked up and down stairs; he sat to table when eating his dinner, the nurse feeding him. At the earlier part of the illness he wished to have the newspaper read to him, but it was doubtful how far he comprehended the subject-matter. Later on he quite failed to take interest in anything, and only afforded a momentary and partial recognition to his relatives. On August the 17th, 1889, he had an attack of syncope. On September the 9th the back became very weak and he could not erect himself as formerly; at this date he ceased to be able to walk. On October the 10th he became cyanotic for a few minutes; when seen two hours after, the pulse was quickened and the cyanosis had disappeared, the patient being very prostrate and in a semi-comatose con-

dition. The following morning the coma had deepened and the pulse was running down. Death took place at 9.45 p.m. on the same day.

The chief medical interest in the case centres in the Cheyne-Stokes breathing and its prolonged duration. I will therefore give a detailed account of the phenomenon :—

The periodic breathing was unaffected by position. Having visited the patient at weekly intervals during the whole time that he suffered from the Cheyne-Stokes breathing, I had abundant opportunity of observation under a variety of conditions, as when lying in bed on his back, on the right and left sides, sitting propped up in bed, sitting on chair, and also standing upright. The character of the phenomena was precisely the same in all cases, allowing for variations in the duration of the phases of which each cycle was composed. Such variations were not dependent on the position of the body, nor on any other external condition, but occurred from time to time in the progress of the disease, and lasted from a few hours, to days or weeks. The following description is from notes taken at the time, after close observation of the symptoms for many weeks. The patient is sat up in bed with the legs resting horizontally before him. The chest is covered with night shirt only, and the lower part of the body and the legs with a sheet and counterpane, so that any movement can readily be seen. The trunk is upright, the arms resting on the bed clothes covering the thighs, but not supporting (or only to a slight extent) the upper part of the body, which is maintained in position partly by pillows placed behind it, and partly by the dorsal muscles. The face is somewhat pallid—not anæmic in the sense of being entirely devoid of colour, but like that of a person who has been confined to his room for a few months. The head is slightly bent forwards, as would be the case if the patient was looking at his feet, the line of sight being at a right angle to the plane of the face. The right side of the face being partially paralysed, displays the corresponding angle of the mouth at a lower level than that of the opposite side; the lips and teeth are separated to a slight extent. The lips are normal in colour. The eyes are open so that the pupils can be clearly seen. The

pupils are about two millimetres in diameter, they are equal, and respond both to light and to accommodation. Taking the cycle in the midst of the respiratory phase, the patient is seen to be breathing in a perfectly normal manner. After a few respirations, the depth of each successively diminishes, and after six more the breathing has entirely ceased. These six downward steps are not equal: if represented graphically, a line drawn along their apices would form a curve, showing that the diminution is proportionally greater in the first three than in the last three. The time occupied by each respiratory act during the downward progress is the same as that required for each act of tranquil respiration, the height of the waves diminishes, but the intervals between them are unaltered. During the pause the face is perfectly still; the eyes remain open, and there is a longer interval than usual between the winking of the lids. The facial expression is peculiar: it is that of a person absorbed in thought, as when the eyes are fixed on an object of which the mind is not cognisant, the brain being entirely occupied in profound cogitation on a subject far removed from the physical surroundings. Such is an exact description of the facial expression, but as a matter of fact no cerebration is taking place. The pupils remain unchanged; in no single instance could I detect any diminution in their size during the pause. The colour of the face, including the lips, is unaltered, there is not the slightest appearance of cyanosis nor of flushing. The ends of the fingers under the nails also retain their colour. If left to himself the patient is usually perfectly still during the pause, but he is not unconscious. If I say—"Well! good morning," and hold out my hand as though to shake hands before leaving, he makes a movement of the hand as one who is wishful to reciprocate the salutation; if requested to put out the tongue he does so. To obtain response, however, it is necessary to utter the words in a louder tone than customary. The movement of the hand is not accompanied by any change of facial expression, nor in the direction of the eyes, as is the case under similar conditions when respiration is taking place. It has all the appearance of an automatic act, the words provoking the movement without the intervention of volition. No

artificial stimulus had any effect on the duration of the pauses. The pulse is not altered neither in rate nor in character during the pause. This was ascertained on many occasions, both by the finger and by the sphygmograph. The duration of the phenomena enabled me to make numerous observations which were carried out with the greatest care, the important bearing of this question being obvious. Tracings were taken during entire cycles, without revealing the slightest alteration, either in rapidity or in tension. There was an occasional irregularity in the pulse-rate—rarely amounting to intermission—but the inequality was limited to individual cardiac contractions and in no instance had it any relation to the respiratory function. After an interval of total cessation of breathing, usually of from 15 to 30 seconds in duration, it is re-established by a series of respirations, of which the first is exceedingly shallow, the second deeper, and so on until the normal depth is slightly exceeded. Then follow three or perhaps four respirations of this slightly exaggerated type, after which the respirations become tranquil and easy until the cycle recommences. The breathing rate during the phase of tranquil respiration was usually about 18 to the minute. During the period of forced breathing the right side of the thorax did not expand so freely as the left. The duration of the period of breathing, taken from the first respiration after a pause to the last which preceded the following pause usually varied from 20 to 42 seconds. The period of breathing did not consist of a series of respirations forming an upward and downward curve, the latter immediately following the former, but the two were separated by a horizontal line representing a phase of regular and equal breathing.

When breathing was resumed, the patient's face lost the look of abstraction, otherwise there was no change. As previously mentioned, the pupils did not contract during the pause, nor was there any change in size on return of breathing. On no occasion did I see the least trace of involuntary movement of the limbs, nor twitching of the mouth when the breathing recommenced, nor was there any rigidity of the muscles of the limbs during the pause. When awake, in addition to voluntary movements of the eyes, the patient

usually made some slight change of position after the breathing was re-established, this was evidently to relieve the irksomeness of the previous fixity of position. The stillness during the pause only occurred when the patient was unoccupied at the moment of cessation of respiration. If he was walking at the time, even going up or down stairs, he continued to do so. It not unfrequently happened, after leaving his room and arriving in the dining room, that he sat down just after the pause commenced; the absence of breathing then appeared even more remarkable than at other times. After exercise the succeeding forced respirations were more energetic than usual and continued during the entire respiratory phase. If the patient was eating he continued to masticate and to swallow the food during the pause just as at other times. On these occasions the facial expression conveyed the impression that the movements, whether of walking or of eating, were non-volitional. At intervals during the last few months of his illness, the patient suffered from attacks of excessive secretion of mucus into the larger bronchial tubes. These attacks came on quite suddenly and without external influences, such as cause bronchial catarrh. They were apparently due to abnormal action of the nerves presiding over the bronchial secretion, and they disappeared as suddenly as they appeared. When this excess of mucus was present, the breathing was much embarrassed. On account of the hemiplegia, together with the general feebleness, the patient had very little power to cough, the mucus consequently accumulated until suffocation seemed imminent. At such times the forced respirations which followed each pause were dyspnœal in character, the expiratory efforts being excessively energetic; they entirely displaced the phase of tranquil breathing and continued until the commencement of the descending steps which led to the succeeding pause. On these occasions the periods of breathing were shorter than usual. The face became distinctly cyanosed, and the patient was much distressed; this he manifested at each re-establishment of breathing by beating the bed clothes with his hands, and by groans and loud cries. During the pauses, when suffering from these bronchial attacks, he remained perfectly

still as at other times, the contrast between the two phases being most remarkable.

The phases of which each cycle was composed were fairly constant for any one period, but they underwent certain changes at various stages of the disease. At the early period of the Cheyne-Stokes breathing there was at times no actual pause, the curve representing the gradual lessening in amplitude of the respirations no sooner approached the base line than it began to rise again. This lasted only a few days, and, with one or two exceptions of not more than two or three hours' duration, did not return. The shortest pause observed by me lasted eight seconds, and the longest thirty-eight seconds. I was assured by the nurse that many times during sleep the pauses lasted much longer than the longest of these two periods, so long that, although she was accustomed to the periodic breathing, she felt convinced on several occasions the patient "had drawn his last breath." The shortest period of breathing observed by me was seventeen seconds, and the longest sixty-one seconds. On one occasion (May 13th, 1889) the breathing was continuous during sleep, but resumed the periodic type when the patient awoke. Again, on August 11th, 1889, the breathing was continuous for several hours both sleeping and waking. With these two exceptions, the Cheyne-Stokes breathing continued from August 19th, 1888, until October 11th,1889, a period of 417 days. About twelve hours before death the periodic breathing ceased and was replaced by continuous laboured breathing, such as frequently precedes death from cerebral disease. This lasted six hours, after which the Cheyne-Stokes breathing was resumed and continued until death took place. About an hour before death the patient yawned repeatedly with extreme energy, so much so that the nurse thought it necessary to support the lower jaw with her hand to prevent dislocation. No treatment, nor drugs, as amyl nitrite, pilocarpine, strychnine, &c., had the least effect on the Cheyne-Stokes breathing. I am indebted to my friend, Dr. Robinson, of Owens College, for the following account of the pathological changes which he found in the brain and medulla.

The most important changes which were noted when the

macroscopical examination of the brain was made related to the arterial system: the vertebral, the basilar, and all the cerebral arteries and their branches had undergone a degeneration which converted them, almost entirely, into calcareous tubes.

The membranes were adherent to each other over the vertex of the brain, and over the same region the dura mater was almost inseparable from the skull-cap, but the membranes were not adherent to the cerebral substance.

Macroscopical sections of the cerebral hemispheres revealed no degenerative changes, but all the cavities of the brain, especially the lateral ventricles, were markedly dilated.

The medulla and the pons were hardened in Müller's fluid, and the former was afterwards embedded in celloidin and cut into sections, from a short distance below the calamus scriptorius up to the acoustic tubercule. Microscopical examination of these sections, after staining by Weigert's and Pal's methods, revealed two distinct abnormalities. First, there was well-marked degeneration of one pyramidal tract; and second, there was very distinct capillary congestion throughout the regions of the root nuclei of the spinal accessory and vagus nerves, the crowding of the corpuscles in the capillaries of these regions contrasting strongly with the absence of any such appearance in the surrounding areas.

Space will only permit mention of a few out of the many theories advanced in explanation of the mechanism of Cheyne-Stokes breathing. They may be divided into two groups; those which are, and those which are not based upon an assumed close correlation between the symptoms and the chemical condition of the blood. Of the first group, the earliest explanation was that of Traube,[1] who postulated a diminished irritability of the respiratory centre from cerebral pressure, or from deficient or deteriorated blood supply. This necessitated more carbon dioxide than in the normal condition to stimulate the centre into activity, and therefore, until the requisite amount accumulated the respiratory centre was inactive. When sufficient carbon dioxide accumulated, the endings of the vagi in the lungs were first stimulated, producing the shallow

[1] *Berlin. klin., Wochenschr.*, 1874.

breathing which immediately follows the pause. Next, the cutaneous nerves were stimulated, causing the stage of laborious breathing. The deep breathing soon relieved the blood of its excess of carbon dioxide, and then the stimulation of the respiratory centre subsided in inverse order—that from the cutaneous nerves first, afterwards that from the vagi. Traube subsequently added exhaustion of the respiratory centre as an additional factor in the causation of the pause.

Filehne[1] amplified this theory by supposing that the vaso-motor centre as well as the respiratory centre is implicated, and that in the normal condition venous blood stimulates first the respiratory centre, and afterwards the vaso-motor centre. He explains the phenomena thus :—the irritability of the respiratory centre is diminshed, and it consequently fails to respond to the stimulus of ordinary venous blood. During the pause the blood increases in venosity, but on account of lessened irritability the respiratory centre is not stimulated into activity until the vaso-motor centre responding to the excessive venosity of the blood produces contraction of the arterioles and so lessens the peripheral blood-supply. Along with other parts the respiratory centre suffers from the anæmia thus caused, and is thereby stimulated into activity. Then follows the shallow breathing which deepens into dyspnœa before the arterioles have time to regain their normal condition and permit the now arterialized blood to reach the medulla. Filehne instances increased arterial tension during, or even before the pause, in corroboration of his hypothesis. Filehne's theory in its turn has been ingeniously amplified by Byrom Bramwell.[2]

We now come to the second group. Mosso[3] regards Cheyne-Stokes breathing merely as an exaggerated form of a physiological phenomenon which may be observed during normal sleep in the human subject, especially in advanced age, and also, as observed by Luciani and Fano,[4] in the sleep of hybernating animals.

[1] *Berlin. klin. Wochenschr.*, 1874.
[2] *Diseases of the Heart and Thoracic Aorta, Edinburgh*, 1884.
[3] *Archiv f. Physiologie.* 1886.
[4] *Lo Sperimentale*, 1879 & 1883.

Rosenbach[1] regards lessened irritability of the central organs of the nervous system especially of the respiratory centre in the medulla, due to defective nutrition from diminished or altered blood-supply, as the causal factor in the production of Cheyne-Stokes breathing. The centres not only have their irritability lowered, but are also more easily exhausted by performance of function. He states that neither artificial respiration, nor the inhalation of oxygen exercise any influence on the periodic breathing.

Quite recently Wertheimer[2] has advanced the hypothesis that the diminished irritability of the respiratory centre, which is the primary cause of Cheyne-Stokes breathing, is due, in some cases, to purely dynamic influence—to an incomplete inhibition. He expresses the opinion that various lesions of the cerebrum, either by nerve-conduction or by modification of the intra-cranial pressure, produce irritation of the medulla, and thus determine Cheyne-Stokes breathing.

In discussing the probable ways in which the respiratory movements may be modified by abnormal influences on the respiratory centre, it will be necessary first to review as concisely as possible the recent physiology of the subject.

According to Marckwald[3] there are in the medulla, in close relation to the nucleus of the vagus, double respiratory centres—an inspiratory, and a less easily excitable expiratory centre, the former of which only is called into action in normal respiration. There are no higher centres for respiration. The respiratory centres in the medulla are both automatically active and reflexly excitable, but, automatically, they can only occasion spasmodic, not rhythmic breathing. Normal rhythmic respiration is a reflex act principally called forth by the intervention of the vagi, which prevent abnormal accumulation of tension in the centres, and convert the inherent irritability of the respiratory centres into regular movements. The vagi have a tonus which probably is sufficient, when the organism is at rest, to regulate the respiratory movements. Next to the vagi, the upper cerebral tracts exercise an important function, and are capable of

[1] *Zeitschr. f. klin. Med.*, 1880. [2] *Archives de Physiologie*, 1890.
[3] *Zeitschr. f. Biologie*, 1886.

replacing the vagi, as they (the vagi) are also capable of compensating for the loss of the upper tracts. If a portion of the upper tracts fail the vagi continuing to act, Cheyne-Stokes breathing may occur. For the production of periodic breathing no alteration in the irritability of the respiratory centre itself is necessary. The centre, either during the period of breathing or during the pause, can be equally excited by similar weak stimuli. The normal irritability of the respiratory centre is not dependent either on want of oxygen, nor on excess of carbon dioxide in the blood. The condition of apnœa has nothing to do with the gaseous contents of the blood, but is probably caused by liberation of the accumulated energy of the respiratory centre by the action of the vagi. During the apnœa neither direct excitation of the medulla, nor of the vagi can produce respiratory movements. On the other hand, Langendorff and Franck,[1] from experimental observations, came to the conclusion that the respiratory centre is not only automatically active, but that it can itself maintain rhythmic breathing, which differs but slightly from ordinary respiration. Loewy[2] made a series of experiments on rabbits to ascertain the influence exercised by the upper brain tracts, the vagi, and other nerves, on the respiratory centre. When the respiratory centre was separated from the higher centres by cutting through the medulla immediately above the respiratory centre, very little alteration was produced in the breathing. When the vagi alone were divided the breathing was slowed and deepened without being materially altered. When both upper tracts and vagi were detached from the respiratory centre a marked change of rhythm occurred. Contrary to Marckwald, but in accord with Langendorff, Loewy holds that the respiratory centre is capable of liberating rhythmic impulses automatically. In two of Loewy's experiments, Cheyne-Stokes breathing occurred after detachment of the upper tract, but when the vagi were subsequently divided the breathing regained the normal rhythm. Wertheimer[3] found on dividing the cord in

[1] *Archiv f. Physiologie*, 1888. [2] *Pflüger's Archiv*, 1888.
[3] *Loc. cit.*

dogs, immediately below the medulla, and then performing artificial respiration until automatic respiration returned, that the breathing was of the periodic type. This points to the existence of sub-centres in the cord below the medulla, which may act automatically. He therefore would not limit the area affected in Cheyne-Stokes breathing to a circumscribed portion of the central nervous system, but would distribute it over the whole extent of the grey axis which governs the respiratory mechanism. He found that Cheyne-Stokes breathing is not necessarily the result of changes in the gaseous condition of the blood, nor need there be any alteration in arterial tension. Some recent experiments of Geppert and Zuntz[1] tend to prove that abnormal balance of oxygen and carbon dioxide in the blood is not the only factor by which it may act as a stimulant to the respiratory centre. By detaching the muscles of a limb in a rabbit from all nerve-paths which normally connect them with the medulla, and then tetanising the muscles, any action produced on the respiratory centre can only be transmitted by the blood. The effect produced was increased respiration, such as results from voluntary active exercise of the muscles. It was found by direct analysis that the tendency to excessive venosity of the blood from the increased muscular activity was more than compensated for by increase in the respiratory acts—that the blood was more aërified than was necessary to maintain the gaseous equivalent that occurs in rest and work under normal conditions. The inference is that certain non-gaseous substances in the blood, resulting from tissue-change, are able directly to stimulate the respiratory centre without the intervention of the endings of the vagi. In relation to this question, Lehmann[2] found by experiments that diminished alkalinity of the blood, caused by the acid products resulting from muscular metabolism, is a very important factor in the stimulation of the respiratory centre. He conjectures that possibly the effect of carbon dioxide on the respiratory centre depends on its acid properties.

It will be seen from some of the experiments that the

[1] *Pflüger's Archiv*, 1888. [2] *Pflüger's Archiv*, 1888.

older view—that of Flourens—which regards the respiratory centre in the medulla as the prime and sole mover in the respiratory act, is no longer tenable. There is abundant evidence to prove that there are sub-centres in the cord below the medulla, and there is reason to believe that these sub-centres are competent to liberate respiratory impulses of modified character without the aid of the medullary centre. As expressed by Langendorff,[1] the automatic centre is not an anatomical, but only a physiological unit, which is made up of the conjoint action of the centres in the medulla and in the cord, its activity being modified by the regulating centres in the medulla. For the present purpose it will be unnecessary to discuss the duplex function of the medullary centres.

Let us now consider by the light of the above-named experiments and others presently to be mentioned, together with the results of my observations on the case narrated, the various hypotheses advanced in explanation of the phenomena of Cheyne-Stokes breathing.

The theories of Traube and Filehne are based on a supposed failure of the respiratory centre to respond to the stimulus of ordinary venous blood, and that an excessive accumulation of carbon dioxide is required to bring it into action. They differ as to the mechanism by which this is effected. Against Traube's theory is its failure to account for the gradual increase in amplitude of the respiratory movements after each pause. If the renewed activity is the result of excess of carbon dioxide in the blood, this stimulus will be at the maximum immediately before the first respiratory movement after a pause. With each subsequent respiration the amount of carbon dioxide would be lessened and its power over the respiratory centre consequently diminished; but notwithstanding this the activity of the centre goes on increasing progressively, and the respirations for a time continue to augment in amplitude. To overcome this objection, Filehne makes the excess of carbon dioxide act on the respiratory centre through the intervention of the vaso-motor centre, and in this way gains time for the gradual

[1] *Arch., f. Physiologie*, 1887.

development of the respiratory movements. One of the principal clinical objections to this theory is the fact that increase in arterial tension—necessitated by the theory—is by no means invariably present in Cheyne-Stokes breathing. Since the theory was formulated a great many cases have been recorded in which no alteration in tension occurred. My case adds another to the list. In many cases in which alteration of tension has been observed, no constant relation existed between the changes in tension and the phases of periodic breathing. In some the tension was greatest at the end of the pause; in others during the phase of the greatest amplitude of respiration, or at the period of maximum dyspnœa. Rosenbach directs attention to the want of accord between the regular alternations of the respiratory phenomena and the capricious behaviour of the circulatory changes, as evidenced by clinical observations. Such discrepancies make it impossible to regard any alteration in arterial tension that may occur as a coefficient in the production of Cheyne-Stokes breathing. Physiological experiments also are against this hypothesis. Sokolow and Luchsinger,[1] and independently of these observers, Langendorff and Siebert,[2] have shown in frogs, that when the circulation through the medulla is prevented, either by clamping the aorta, or by rapid bleeding, periodic respiration is produced. If after clamping the aorta and production of periodic breathing the clamp is removed, the normal type of respiration returns after a varying period, which has a direct relation to the length of time that the circulation through the aorta was interrupted. All these observers agree in attributing the periodic breathing to exhaustion of the respiratory centre. Filehne suggests that if exhaustion is the cause it ought to assert itself after each individual act of respiration. Luchsinger's explanation is, that the irritability of a nerve centre increases when after rest it is again stimulated, and diminishes if the period of activity is prolonged. Although the stimulation may be continuous, the effect of the stimulation may manifest itself in periodic impulses. In illustration of

[1] *Pflüger's Archiv*, 1883.
[2] *Archiv f. Physiologie*, 1881.

this he cites the experiment by Stirling[1] of administering weak rhythmic induction waves to the foot of a frog, by which a series of increasing and decreasing contractions followed by a pause were obtained. Langendorff maintains that the exhaustion itself is solely antagonistic to the renewal of rhythmic breathing, and that its disappearance is gradual. Therefore the first movement after a pause being still under the influence of the subsiding exhaustion, is feeble. Each succeeding breath is further removed from the antagonistic influence, and consequently increases in depth until full amplitude of movement is attained. The movements then diminish, because the energy expended is greatly in excess of the supply. In another experiment, Sokolow and Luchsinger measured the blood-pressure in a frog by means of a monometer, after the production of Cheyne-Stokes breathing by clamping the aorta (the aorta being subsequently released), without any material change being observed; the integrity of the vaso-motor apparatus was demonstrated by stimulation of the sensory nerves, which occasioned marked increase in the blood-pressure.

In my case, it is to be noted that when the patient was in his ordinary condition there was no appearance of cyanosis during the pauses, however long they lasted. When the gaseous interchange in the pulmonary air-vesicles was impeded by excess of mucus in the bronchial tubes, however, there was marked and continuous cyanosis, and during each period of breathing the patient displayed all the anxiety of one suffering from impending asphyxia. Granting that diminished irritability of the respiratory centre is the cause of the cessation of breathing, and that excess of carbon dioxide is the cause of its periodic renewal, the pauses ought to have been abolished so long as this state of cyanosis lasted, since the venosity of the blood then existing was undoubtedly greatly in excess of that which occurred in any period of respiration when no excess of mucus was present. If the amount of carbon dioxide in the blood during the ordinary course of the Cheyne-Stokes breathing was sufficient to stimulate the

[1] *Sächsische Berichte*, 1874.

sluggish centre for a time, the vastly increased amount under the conditions indicated would have been sufficient to have kept it in activity as long as the excess continued. It may be urged that the respiratory centre adapted itself to the new state of affairs, its irritability being further reduced, so that its relation to the increased venosity of the blood was the same as before, the entire level at which the phenomena took place being lowered. There is a limit, however, to the depression in irritability of centres primarily concerned in the performance of functions necessary to the maintenance of life. Beyond a certain point it is no longer a question of susceptibility to the action of stimuli only that is concerned; that is simply one manifestation of an essential condition—the capacity of the elements involved to liberate energy. When the irritability of the respiratory centre is excessively lowered by faulty nutrition, so is its energising capacity—the former being the outcome of the latter, or rather, both are dependent on the same molecular activity. If the molecular activity is lowered to such a degree as to necessitate an ever-increasing amount of stimulus to compel liberation of energy, it constitutes proof of a progressively diminishing capacity of the cells implicated to yield energy. When the bronchial secretion was in excess, the venosity of the blood was so great that, had it been simply a question of reduced irritability of the respiratory centre—to any degree compatible with retention of molecular activity equal to performance of function—it must have been stimulated into continuous activity. If, as suggested, the level of the phenomena had been lowered proportionately to the excessive venosity of the blood during the bronchial attacks, the energising power of the respiratory centres would have been incompetent to liberate a series of effective impulses; the most that they could have done would have been to yield detached impulses at irregular intervals—the outbursts of final exhaustion—and that for a short time only. A further objection is, that depression in irritability such as enables nerve centres to accommodate themselves to altered external relations is of gradual development, and is not suddenly produced by excess of stimulus. The effects of exhaustion

from over-stimulation do not at present enter into consideration.

The case itself afforded further and positive proof that the immediately antecedent condition of the blood was not concerned in the production of the Cheyne-Stokes breathing. When a pause occurred whilst the patient was walking from one room to the other (the bronchial secretion being normal), the breathing during the subsequent period of respiration was dyspnœal throughout, the excessive amplitude of movement not being limited as under ordinary conditions to the first two or three breaths. This points to actual stimulation of the respiratory centre, probably to be explained by the light of the previously-mentioned experiments of Zuntz and Geppert, as resulting from the non-gaseous products of muscular activity, there being no appearance of cyanosis on such occasions. Nevertheless, there was no interruption to the Cheyne-Stokes breathing. Although evidently undergoing excessive stimulation and responding to it, the respiratory centre ceased to do so in the midst of the excitation. When the patient sat down further development of these respiratory stimuli from muscular movements ceased, but their elimination was not completed during the following period of breathing; it was resumed after the first, and sometimes after the second pause, in the succeeding respiratory phases. Under these circumstances, there is no time for the respiratory centre to adapt itself to altered conditions, the stimulus is developed in a few seconds, and produces an immediate effect. Further development is suddenly arrested. The limited period of respiration which follows is insufficient for the elimination of the stimulus, and instead of the period being prolonged until all excess is removed, and the balance between the respiratory centre and the blood restored (as would be the case if the intermission depended on ineffective stimulation of the centre), the completion of the task is held over till after the next pause.

It is obvious that a causation other than the chemical condition of the blood must be sought for, and the attention is naturally directed to changes in the respiratory centre itself. In the normal condition we may suppose that the

nerve cells concerned in the production of the respiratory movements convert the oxygen and other substances contained in arterial blood, which are spoken of as nutritive elements, into energy, and within certain limits store it up. In proper course this energy is transformed into work. Two intrinsic conditions may affect prejudicially the quality of the work done :—increased resistance to liberation of energy present in normal amount, and diminished amount of energy, the resistance to discharge being unaltered. In relation to the phenomena under consideration, the first of these conditions might result from inhibition, acting on the respiratory centres in such a way as to interfere with their normal discharging potential. It is quite conceivable that such inhibition would be effective if it directly influenced the respiratory centres in the medulla. Physiological experiments show that electrical, mechanical, and chemical stimuli directly applied to the medulla are capable of causing inhibition of the respiratory centres. Some recent experiments by Unverricht[1] tend to demonstrate the presence in the cortex of filaments (an actual inhibitory centre is not claimed) which, when stimulated, exercise an inhibitory influence over the respiratory centre. By means of a carefully graduated faradic current applied to a limited area of the cortex, Unverricht prolonged the duration of the expiratory pause. How inhibition can be produced by pathological changes, however, is not clear. If by inhibition we mean arrest of function in a centre, produced by a restraining influence extrinsic to it, the influence thus exercised acts in antagonism to the centre restrained. When effective, this implies greater energising power on the part of the controlling mechanism than on the part of that which is controlled. It is difficult to account for such a dominating influence being developed in any part of the higher cerebral tracts—its assumed locality—when they, as well as the respiratory centres, are labouring under the same defective nutrition. In the causation of Cheyne-Stokes breathing inhibition would need to be rhythmic ; if it were constant another factor would be necessary to account for the alternate gradual

[1] *Verhandlungen des Congresses für innere Medicin*, 1888.

rise and fall in the respiratory movements, either the immediate effect of changes in the chemical condition of the blood, or periodic overflows of energy from the cells inhibited. The former agency has already been discussed; the latter is a more complicated way of expressing fluctuations of energy at a higher level, which are about to be discussed under simpler conditions. The hypothesis of rhythmic inhibition would only remove causation a step back; the periodic nature of the phenomena would remain unsolved. In addition to difficulties in the way of accounting for the mode of production of inhibition, there are doubts as to its competence to produce the phenomena observed in the above reported case. It is questionable how far the respiratory centre, when under the influence of inhibition, can resist the powerful stimulus of excessively venous blood. In relation to this subject some of Head's[1] experiments are extremely suggestive. In producing apnœa by artificial respiration, Head found that as far as the advent of apnœa is concerned, it is immaterial whether the inflations are made with air, oxygen, or hydrogen, there is no observable difference. But there is a marked difference as to the several durations of the apnœa thus produced. With hydrogen the apnœa pause is short, and often the rhythmic breathing begins again immediately after cessation of the artificial ventilation. With air the pause is longer, and with oxygen the longest of all. The apnœa is not due to excess of oxygen in the blood, but to summation in the respiratory centre of the stimuli, caused by the artificially-produced periodic alterations in the volume of the lungs—a form of inhibition. The apnœa is sooner or later cut short by the advent of dyspnœa, which overcomes the after reaction of the inflations, and determines the return of rhythmic breathing. The interesting point is:—that the dynamic effect of the three gases is the same; the number of inflations required to produce the apnœa is identical in all cases. Starting with the same degree of inhibitory influence, we find that the rapid occurrence of dyspnœa, when hydrogen is used as inflating medium, quickly breaks down the inhibition and determines rhythmic respiration; whereas when

[1] *Journal of Physiology*, Vol. x.

oxygen is used the advent of dyspnœa is postponed, and the inhibitory influence exercises for a time undisputed control. I do not think in my case that, when the cynanosis was present, any stimulus could have determined an inhibition sufficiently dominant as to have rendered the respiratory centre insensitive to the exceedingly venous condition of the blood then existing.

The second of the two conditions postulated as influencing the quality of the work done by the respiratory centres —diminished amount of energy—remains to be considered. This hypothesis is in accord with the observations of most of the physiologists whose experiments have been mentioned and certainly affords the most consistent explanation of the phenomena which occurred in the case I have recorded. The progress of the case during life and the appearances after death, indicated clearly that interference with the blood supply to the central nervous system was the cause of the train of symptoms which occurred. The arteries supplying the brain and medulla were the seat of degenerative changes which had been in progress for many years. As the arteries became gradually obstructed, the nutrition of the dependent nerve-cells was progressively lowered, and they underwent slowly advancing molecular changes. The psychical powers were the first to succumb; the mental capacity, originally of the highest type, gradually faded. It is to be observed that the alteration was purely quantitive—simply a progressive diminution in intellectual energy without aberration. The first indication was, that problems which the patient formerly would have quickly solved on the spot, had to be reserved for deliberation. After a time the mind was unable to grasp the premises of an argument involving much co-ordinative power, and eventually it failed to comprehend isolated facts. Before this last stage was reached the physical powers were involved. Eventually the cell-groups concerned in the innervation of the respiratory apparatus suffered from the effects of the slowly increasing mal-nutrition, the result being a diminution in their energising power. As the action of the respiratory centre is probably automatic, apart from modification by external influences, voluntary and reflex, it would seem (in

response to some stimulus with the nature of which we are at present unacquainted) to be capable of liberating rhythmic impulses, each impulse being independent of the immediately antecedent condition of the blood, being simply the equation of the molecular activity by which the nerve-cells are energised. Under normal conditions there is probably stored up in the nerve-cells a reserve of potential energy that places the respiratory movements out of the reach of immediate want, so that a temporary slight derangement of nutrition is not attended by alteration of function. Serious disturbance of metabolism, however, resulting either from prolonged malnutrition, or from some interference of recent origin, lowers the activity of the cells, so that they are unable to maintain any reserve of energy; production and consumption then go on side by side. A very slight further reduction places the balance to the wrong side of the account, and continuous rhythmic respiration can no longer be maintained. As the available energy sinks, so do the respiratory impulses until the pause is reached. For a time there is no output, and during the early part of the pause the cells are probably quiescent. They then gradually resume activity until the energy developed reaches a certain potential, when discharge of impulses re-commences. This initiates the shallow breathing. The activity of the nerve-cells is still increasing and continues to do so for some time after the discharging point is reached, and as a consequence the impulses become stronger and the respiratory movements gain in amplitude. In the case narrated, when excess of bronchial mucus was present, there was, as stated, distinct cyanosis with pronounced air-hunger during the respiratory phases. At these times the undoubted excess of carbon dioxide and absence of oxygen in the blood, entirely failed to stimulate the respiratory centres into activity during the pauses; but on each occasion when breathing was re-established, the stimulus brought to bear by this condition of the blood exercised a powerful influence on them. This influence however, did not produce dyspnœal respiration immediately after re-commencement of breathing; the step by step development in amplitude of movements took

place as usual. These two facts have an important bearing as regards the interpretation just given of the mechanism of Cheyne-Stokes breathing. First, during the pause the excessively venous blood was unable to stimulate the respiratory centres into activity; second, when breathing had re-commenced it was equally unable to produce dyspnœal breathing until the centres had regained their full activity. It is evident that the same cause was at work in both instances. The hypothesis advanced assumes the cause to be exhaustion of the energising centres—complete in the first instance, and partial in the second. The absence of response was not due to blunted sensitiveness to stimulation, but to a more radical defect—want of energy, the cells were run down. Whilst the impulses increased step by step, the energy thus liberated was all that was being developed at the moment and it consequently could not be augmented by a stimulus. Stimulation can only liberate energy if it is present, or, if not potentially pre-existent, by developing the energising capacity of the cells concerned in its production. During the ascending group of respiratory movements the cells were doing the utmost permitted by their then state of activity—stimulation could not make them do more; but when their energising capacity was again in full swing, stimulation could, and did cause increased liberation of impulses, manifested by excessive amplitude in the respiratory movements, which continued until the first of the descending group leading down to the next pause, not as under ordinary circumstances, being limited to the first three or four of the fully developed movements. The natural result was more rapid exhaustion of the energising cells than usual; the periods of breathing being shorter under these conditions than when the bronchial secretion was in normal amount. It is possible that summation of stimuli in the respiratory centre, conveyed by the vagi, resulting from the excessive amplitude in lung movements, might aid in determining the earlier arrival of the pause. The three or four respirations of excessive amplitude, described as occurring after each pause under ordinary conditions, when the patient was free from the bronchial trouble, were probably, in some measure due to

slight excess in venosity of the blood, the balance being speedily re-adjusted.

The absence of distress during the pauses requires explanation. It may be accounted for either by supposing that the upper brain-tracts participated in the periodic exhaustion, or that exhaustion of the respiratory centres alone was sufficient to abolish for a time the sensation of air-hunger. The permanence of the psychical and to a lesser extent of the motor symptoms, indicates that the upper brain-tracts had undergone some amount of actual structural change. As regards the hemiplegia it is to be observed that although no changes were found in the hemisphere, the degeneration in the pyramidal tract points to antecedent destruction of some of the cortical cells or of their filaments. In addition to this, malnutrition would lower the activity of the structurally unchanged cortical cells and they would consequently readily succumb to exhaustion. That a condition of partial exhaustion of the sensorium periodically occurred is probable, but it is doubtful if it was to a degree sufficient to render the patient insentient to the dyspnœa. When the bronchial secretion was in excess the absolute indifference of the patient (not due to general insensibility) to conditions which only a few seconds previously had evoked the most acute manifestations of distress, indicated complete interruption of the stimuli which provoked them. I am therefore disposed to think that periodic exhaustion of the respiratory centres may have been the cause of abolition of the perception of dyspnœa. Hein,[1] Sacchi,[2] Ewald,[3] and other observers have noticed a condition of drowsiness during the pause, and in some of these cases the patients were sufficiently conscious during the respiratory phases as to explain that they felt no discomfort during the pauses, but that they suffered greatly from dyspnœa with each return of respiration, the distress beginning with the first shallow breath. In my patient there was no actual drowsiness, merely a state of passivity, with absence of perception of dyspnœa. His mental state and the aphasia, prevented

[1] *Deutsch. Arch. f. klin., Med.*, 1880.
[2] *Rivista clinica di Bologna*, 1877.
[3] *Berl. klin., Wochenschr.*, 1874.

any verbal expression of feeling, but his actions left no room for doubt as to what he did, and what he did not feel. It was most remarkable to see the patient in the midst of anxious manifestations of urgent dyspnœa, on some occasions of almost asphyxial violence, gradually tone down to a condition of perfect placidity with each successive step towards the pause. The return of the perception of the dyspnœa was simply an inversion of its loss. The first manifestation was synchronous with the first shallow breath, as the breathing increased in depth, so did the manifestations of distress.

The changes alluded to in the nerve-cells as occasioned by defective nutrition are probably of a purely molecular character, producing no alteration in the physical appearance of the cells, at least none that we can recognise. It is doubtful whether the microscopic changes found in the medulla bore any direct relation to the Cheyne-Stokes breathing. Influences of a much more subtle nature are at work, which, leaving the cellular framework unchanged, affect only the chemical processes taking place within the cell by which the oxygen of the blood is utilized as a mode of motion, so that the physiological function of the cell is interfered with apart from its existence as an anatomical unit. Imperfect metabolism would lead to this, both by diminishing the supply of oxygen to the cells, and by defective removal of effete products from them. In this way the normal activity of the chemical processes going on in the cells would be damped, and the sum of the resultant energy would be proportionally lessened. It is competent for results of this kind to follow quite recent blood-changes, whether quantitative or qualitative, and also after prolonged existence to quickly give place to renewal of normal function. In my case on three occasions at many months interval, the periodic breathing was replaced for some hours together by normal breathing. On one of these occasions there were alternations of periodic and regular breathing of several hours' duration, in accordance as to whether the patient was asleep or awake. West[1] relates a case in which Cheyne-Stokes breathing, unaccompanied by changes in the pulse or in the pupils during the

[1] *The Lancet*. 1890, p. 545.

pauses, extended over a period of three months. During that time it was replaced on one occasion by simple increase and decrease in the amplitude of the respirations without any pause, and on another by intermissions of two to four respirations, which replaced the pauses. These alternations are most easily accounted for by the hypothesis of exhaustion of the respiratory centres. The difference between fully developed Cheyne-Stokes breathing with pauses, and mere ebbing and flowing of the respiratory movements without pause, is due to the respective states of activity of the nerve-cells. In both instances their energising capacity was sub-normal; in one it was so much so as to necessitate a period of rest for recuperation; in the other, though below what was necessary to maintain regular respiration, it was sufficient to avert an actual standstill. A similar condition, it will be remembered, occurred in the early stage of my own case. The intermissions in West's case would be most likely due to slight depression in the energising capacity of the respiratory centres, with probably some altered sensitiveness to stimuli. The marvel is not that Cheyne-Stokes breathing sometimes undergoes modification in character, but rather that its phases are so regular in their sequence. This is the more remarkable when we consider that the abnormality of function is not dependent upon structural modification of the nerve elements concerned, but upon an altered state of their nutrition. The exceptional duration of my case makes it an extremely interesting example of perverted function without correlative tissue-change, apart from that affecting the vascular supply. The uncertain basis on which the Cheyne-Stokes breathing rested was evinced by the sudden reversions to normal respiration which occurred at protracted intervals, without any ascertainable cause, but presumably from a temporary improvement in the nutrition of the respiratory centres.

A feature of extreme interest in West's case is that when the patient during a pause was asked to take a breath, he only succeeded in setting in action some of the extra-respiratory muscles — the diaphragm and intercostals remained motionless. This limits the enervation to the ordinary

(automatic) respiratory muscles, and indicates that volitional impulses tending to modify the respiratory movements act on, or through, the respiratory centre as co-ordinating centre. Admitting the hypothesis of exhaustion, it is easy to conceive that the volitional impulses were arrested at the dormant respiratory centre (probably in the medulla) and consequently failed to reach the spinal motor nerve-roots.

TREATMENT BY SUSPENSION.

BY J. S. R. RUSSELL, M.B., AND JAMES TAYLOR, M.A., M.B.

In 1883, Dr. Motschoutkowski, of Odessa, published in the *Wratsch* a paper in which he detailed the results of treatment by suspension of several cases of disease of the nervous system—results sufficiently startling, especially as regards the cases of tabes dorsalis, to have merited something more than the scant notice they received at the time. The treatment, however, does not seem to have been practised by anyone until it was brought under Charcot's notice by a medical man, who, during a visit to Russia, had seen it carried out there. In the end of 1888, Charcot gave the treatment a trial, and in the beginning of last year, at one of his *cliniques* at the Salpetrière, detailed his results. Although those were by no means to be compared with what the Russian physician obtained, still, backed by the great name of Charcot, they were such as to attract universal attention, and to ensure for the treatment a thorough trial. We have now had the experience of a year, and it may be well to review the results, in order to see whether the early promise of the treatment has been realized.

As to the manner of carrying out the treatment, Charcot gave directions—and all who have since practised it seem to have had these for their guide—that it was to be done with an ordinary Sayre's apparatus, using both head and arm pieces. The time allowed for the first suspension was to be half a minute, and each subsequent suspension was to be increased by the same period until a maximum of four minutes was reached. A direction was also given that the patient was to raise his arm every fifteen or twenty seconds, in order that the weight of the trunk might be thrown more and more on the neck alone. Suspension was to be carried out on alter-

nate days, and in thirty suspensions all the good likely to result was supposed to have been obtained.

Motschoutkowski, on the other hand, using the same apparatus and the same method generally, had suspended patients for a much longer time. Ten to twelve minutes was not an unusual duration, and five minutes seem to have been about the minimum after the treatment was fairly commenced. The course, too, was a much longer one, 97, 80, and 50 being some of the figures met with in his cases. Emphysema, phthisis, degenerated arteries, were to be regarded as contra-indications, and cases in which laryngeal crises occurred were regarded as unsuitable.

Motschoutkowski's list, as given in a translation of his paper in a recent number of BRAIN (Oct., 1889), comprised in all nineteen cases, twelve being cases of tabes, the others including cases of lateral sclerosis, chronic myelitis, disseminated sclerosis, and sciatica. There was no improvement except in tabetics. Of the twelve all improved except two, and those were early cases. The improvement consisted for the most part in disappearance of the paræsthesiæ and in diminution of the ataxy, but in three of the cases the improvement was remarkable. In the first there had been weakness and muscular wasting, reduced faradaic irritability of muscles, analgesia in the feet, ataxic gait, and unsteadiness in standing. After ninety-seven suspensions there was marked improvement — disappearance of paræsthesiæ, increased muscular force, and less ataxy. The second case was similar, but the change even more remarkable. He was confined to bed and unable to walk. After seven suspensions he was able to get up, after eleven he began to walk, and the improvement continued to advance as the suspensions went on. In a third case there was great weakness, muscular wasting, reaction of degeneration in the muscles, no impaired sensibility, but excessive pain. The pupil reflex was normal. After a long course of suspension, the muscles had regained their contractility, the pains had disappeared, he was able to walk, and the recovery was both complete and permanent. Such a case as this—especially as the patient was a publican—naturally suggests the picture of

multiple neuritis, and without the slightest attempt to question Motschoutkowski's diagnosis, it is well to bear in mind that such cases sometimes deceive the most experienced observers.

In none of the cases was there a return of knee-jerk, or any change in the visual apparatus.

None of Charcot's cases seem to have been so advanced as those just described. In March[1] he had treated in all one hundred and fourteen cases. In sixty-four the treatment was not sufficiently prolonged. Of the remaining fifty, thirty-eight improved, the gait was less ataxic—especially after each suspension, but after eight or ten continuously so—there was greater steadiness in standing, the bladder troubles improved, and so did the pains. There was no change in the condition of the reflexes. In one case out of three of Friedreich's disease, some improvement is said to have taken place. One case of disseminated sclerosis became worse; two cases of sexual failure improved. In eight cases of spastic paraplegia rigidity is said to have become less. Four cases of paralysis agitans are said to have felt better, and to have slept better; but no change occurred in the tremor.

Such results as those were quite sufficient to force this method of treatment under the notice of physicians, and we find lists of cases with detailed results scattered through the periodical literature of the last year. We propose now to refer to those results so far as they have come under our notice.

Morton[2] gives details of six cases of tabes. His best case shewed great improvement in walking, almost total cessation of lightning pains, return of pupil re-action to light, and return of sexual power. Optic atrophy, which was present, became no worse. There was similar if not so marked improvement in the other cases. Five minutes was the time allowed for suspension, and if the arms were not affected, the patient was allowed to suspend himself.

In the same journal Dana gives his experience of the

[1] *Progrès Médical*, June 22nd, 1889.
[2] *New York Medical Record*, April 13th, 1889.

treatment in six cases of tabes. He noted improvement in two—one after sixteen suspensions—the other after ten, no improvement in the others. Two cases of paralysis agitans are said to have felt better, nothing is said as to any change in the tremor. His general impression of suspension is that "it is a measure of minor and occasional value," and after making this statement, he somewhat inconsequently goes on to suggest suspension by the feet, instead of in the orthodox manner.

Simpson[1] reports two cases. One was confined to bed but after a course of suspensions was "able to walk," but the pains were still present: the other experienced improvement in pains and subjective feelings.

Watzfelder[2] has five cases of tabes. Those were suspended at first thrice daily for three minutes at a time, owing to a misunderstanding of the method. After five days of this somewhat heroic treatment, they were reduced to the ordinary alternate day treatment. They are said all to have shewn improvement—the gait becoming less ataxic, the incontinence less marked, and the pains less severe, although the improvement in the last respect was not constant. In one case "diplopia almost gone" is noted.

Eulenberg and Mendel[3] suspended thirty-four cases. In three the improvement was marked—consisting in greater control over bladder, diminution of pains, improved gait and increased steadiness in standing. One shewed a decrease in pains and paræsthesia alone. In none of the three noted as markedly improved, is there complete recovery from any one of those symptoms. In the majority of the remaining cases some improvement was present, affecting bladder function, gait, power of standing steadily, or pains, in four there was no improvement, one improved at first, relapsed later, and one was worse, having distinctly greater trouble with his bladder.

Ladame[4] relates his experience of fifteen cases of tabes, and one of Friedreich's disease. In two there was no im-

[1] *Canadian Practitioner*, June 1st, 1889.
[2] *New York Medical Record*, May, 1889.
[3] *Neurologisches Centralblatt*, June 1st, 1889.
[4] *Revue Médicale de la Suisse Romande*, June 20th, 1889.

provement; in the others there was slight improvement, similar to that experienced in the last series of cases. In two cases there was a return of the power of erection. No complete recovery of control over bladder is claimed. In his experience there was first a rapid improvement followed by a relapse, this being succeeded by a period of gradual improvement attaining its maximum after twenty suspensions.

Balaban[1] working under Dujardin-Beaumetz suspended nine cases. In one there was disappearance of pains, in four they were diminished, in two they disappeared but returned, in one they were not affected. The gait improved in eight, in the other a temporary improvement was succeeded by a relapse. In the same patient diplopia became worse; in one vision is said to have improved.

Haushalter and Adam[2] treated a number of cases suffering from the most varied nervous diseases including tabes, myelitis, neuralgic pains, hypochondriasis, &c., but as we have not yet sufficient data to discuss the effect in such diverse cases, we shall content ourselves with looking at their results in cases of tabes and spastic paraplegia. Of the latter condition they give two cases. No benefit was experienced. Of tabes they report six cases—two, early cases with pains, did not improve. The other four improved, two of them very distinctly as regards gait, pains, and steadiness; in one gastric crises persisted.

Bianchetti[3] details eight cases. In three the treatment could not be continued on account of faintness and amaurosis; in four of the remaining cases the results are described as wonderful, lightning pains, gastric and vesical crises, disappearing as if by magic. He produced no effect in some cases of paralysis agitans and impotence.

Marina[4] suspended twenty-one cases of tabes. In one-half there was marked relief, the remaining half became worse. In one case, a return of the knee-jerk—a violent jerk after five or six taps—is claimed to have occurred.

[1] *These de Paris, Progrès Médicale*, Nov., 1889.
[2] *Progrès Médicale*, Nov. 2nd, 1889.
[3] Quoted in *British Medical Journal*, 1889, vol. ii., p. 938.
[4] Quoted in *British Medical Journal, loc. cit.*

Michell Clarke[1] gives the results which he obtained in fourteen cases—twelve of tabes, one of Friedreich's disease, and one of doubtful nature. He got improvement in ten out of the twelve. The two who did not improve had ocular symptoms. The case of Friedreich's disease experienced no permanent benefit. Briefly, his conclusions are that this treatment increases the power of walking and standing. The pains usually become less after fifteen suspensions; relief of gastric crises is less certain, while knee-jerk, Argyll-Robertson pupils, vision and ocular paralysis are unaltered. It is to be noted that some of this observer's patients received a very long course of treatment, sixty, seventy, and eighty being some of the numbers met with, yet it does not appear that the results were much better than those met with in cases where thirty, twenty, or even twelve were the numbers.

Darier[2] suspended four cases in which optic atrophy was the leading symptom, and in all of them he reports improvement in vision.

Grainger Stewart[3] suspended five cases—three of tabes, two of spastic paraplegia. In all the improvement was marked. The pains disappeared, steadiness increased, and in one sexual power was regained. Two of the cases had only four suspensions, but the effect seems to have been equally great.

Bernhardt[4] suspended nineteen cases. He carried out the treatment every other day, and as he found only one patient who could bear suspension for four minutes, he made three minutes his maximum. In those cases there was diminution and occasional cessation of pains during the treatment; some are described as enthusiasts claiming wonderful improvement in walking while there was nothing objective corresponding to this fancy; one had erections return, with involuntary emissions so troublesome, that he begged the treatment might be discontinued. This observer evidently regards the treatment by suspension as being indebted for what good it does to the mental effect.

[1] *Practitioner*, Nov., 1889.
[2] *Recueil d'Ophthalmologie*, Nov. 4th, 1889, p. 249.
[3] *Edinburgh Medical Journal*, Jan., 1890.
[4] *Berlin Klin. Woch.* June 17th, 1889.

Erb[1] reports six cases. One became a little better, one at first better, later became worse, two were distinctly worse, and one died of an inter-current complication.

Dujardin Beaumetz[2] had twenty cases. In the majority at first some improvement, later a relapse to the former condition; in the minority no change. He is inclined to doubt the value of the treatment.

In the three cases recorded by Hale White,[3] one with pyrexia following suspension, the patients were apparently pleased with the treatment, although he notes that while in two the gait was rather better, and the ataxy less, in one of them pains and gastric crises became worse, and in the third, while the sensory symptoms were rather improved, the ataxy was worse and the other symptoms unaltered. One case had ten suspensions, treatment being stopped on account of the pyrexia. In the other two, the numbers were fifty and twenty-eight, while the time was gradually increased to seven minutes.

Dr. Althaus[4] mentions two cases of tabes, in one of which lightning pains ceased, while gastric crises did so in the other. In one case of paralysis agitans the tremor is said to have ceased for thirty-six hours after the first suspension. The same authority (*Brit. Med. Jour.*, Vol. ii., p. 872) reports a case in which ability to walk was restored, and the knee-jerk returned, but such a case is so remarkable as to make a much more detailed account of it desirable than is given simply in a letter to the Journal.

Dr. Saundby[5] has tried the treatment in six cases of tabes; and we cannot do better than quote his own words with reference to the effect of treatment. He says "It cannot be stated that any one of those cases derived benefit from suspension. One case certainly improved, but he was taking three-grain doses of iodide of potassium, three times daily, and he was also blistered down his spine."

Mr. Lunn[6] reports five cases, one of whom improved

[1] *Neurolog. Centralblatt*, July 1st, 1869.
[2] *Therapeutic Gazette*, 1889.
[3] *Lancet*, Jan. 4th, 1890.
[4] *British Medical Journal*, 1889, Vol. i., p. 429.
[5] *British Medical Journal*, 1889, Vol. ii., p. 692.
[6] *British Medical Journal*, 1889, Vol. ii., p. 765.

considerably in walking power. He was taking iodide of potassium three times a day in fifteen-grain doses. The others remained *in statu quo*.

Churton, of Leeds,[1] suspended only one case, which became worse. He began with suspension for five minutes. In the communication in which he reports this case, he refers to another, which, while waiting for the suspension apparatus, improved so much as to be able to leave the hospital.

Such is the literature of suspension, so far as it has come under our notice. It will be observed that, although there is great variety both in the number and duration of the suspensions, the improvement claimed is fairly uniform. We have suspensions persisted in till eighty and ninety have been given, and improvement results; we have in some cases improvement almost equally well marked with three or four. So also as to the duration of the individual suspensions. While in some cases this is prolonged to ten or twelve minutes, in other patients it is found that three minutes is the longest that can be borne; while in still others three minutes three times a day is given, but whether it is given three times a day or three times a week, the results seem equally satisfactory.

We shall now proceed to lay before you the results of the suspension treatment as carried out by us at the National Hospital, Queen Square, under the direction of the physicians there. As a rule, no difficulty has been experienced in carrying it out. The majority of patients bear it well, and suffer little or no inconvenience; but the toleration varies considerably, and some patients always suffer more or less. In one of our cases nausea and vomiting, or a tendency to vomit occurred in one case during each of the four suspensions he had. Several had slight faintness with dimness of vision; a good many complained of some pain at the back of the neck, or in the jaws after suspension. Two had very severe pain at the lower part of the back at the conclusion of each suspension, and this remained present on each occasion. Other observers seem to have had similar experiences.

[1] *Brit. Med. Jour.*, 1889, Vol. ii., p. 818.

We have had, fortunately, no serious accident, but that the treatment is not without danger is evidenced by the fact that suspension has had already some victims. Two patients[1] were strangled from slipping of the chin-strap; a case is reported by Dr. E. Bloch[2] in which the patient suspending himself against the doctor's orders was killed; a similar accident occurred in a case reported by Dr. Gorecki. Another fatal accident was that which occurred to a medical man in America[3] suspending himself, apparently for experimental purposes. Dr. Borsari, of Modena,[4] reports a case in which serious symptoms came on in a patient after two or three suspensions of fifteen minutes each, and who died soon after of cerebro-spinal meningitis. It will be observed that in none of those cases were Charcot's directions closely followed.

In thirty-seven of our cases the treatment was carried out exactly as Charcot directed—viz., on alternate days, with a maximum of four minutes. In the other eight, suspension was carried out every day, with a maximum of three minutes' duration. The numbers of suspensions were as follows:—

12 had less than 20.
11 ,, between 20 and 30.
22 ,, over 30. Largest number was 50.

The cases were:—

Tabes...	32
Paralysis agitans	5
Ataxic paraplegia	2
Spastic	1
Functional	1
Neurasthenia	1
Doubtful nature	1

Of the forty-five cases nine improved; of these six were tabes, one was paralysis agitans, one was functional paraplegia, and one was of doubtful nature. Five became distinctly worse; the rest remained *in statu quo*.

[1] *Bulletin Médicale. Lancet*, p. 727., June. 1889.
[2] *Progrès Médicale*, 1889.
[3] *New York Med. Jour.*, June 11th, 1889.
[4] *Brit. Med. Jour.*, Vol. ii., 1889.

We shall now consider the cases which improved and those which became worse, with reference to the manner and degree of such improvement or deterioration.

Of the cases of tabes which improved, three were cases in which the improvement, although not great, was distinct and steady, consisting in a less ataxic gait and greater steadiness in standing. One remained subject to occasional lightning pains; another, although better, was still suffering from pain in the loins. Of the remaining three cases noted as improved, one was a very ataxic patient, who had first of all twenty-five suspensions without any change occurring. Soon after this, while having a course of baths in the hospital, he became much more ataxic, and suspension was resumed. After ten suspensions a steady and gradual improvement set in, and when he left hospital, after having in all forty-five suspensions, he was able to walk rather better than on admission. Considering the negative effect of the first course of suspensions, the improvement occurring during the second can scarcely be regarded as anything more than a coincidence. The other two cases of improvement experienced this to a considerable extent at first, having greater steadiness in standing and walking, but towards the end of the course of suspensions—in one of forty-one suspensions and in the other of thirty—they relapsed into their former condition.

So much for improvement occurring in cases of tabes. The other cases which improved were a case of paralysis agitans, a case of functional paraplegia, and one of doubtful nature. In the patient suffering from paralysis agitans, the improvement consisted of abolition of the tremor, which, however, he stated, occasionally returned at home after it had ceased to be present on the occasion of his visits to the hospital. The gait and the other conditions characteristic of the disease remained unchanged. The case of functional paraplegia improved a little, but as this is one of those cases in which one can confidently predict improvements with vigorous treatment of any kind, the fact that she did improve can scarcely be looked upon as shewing that there is any peculiar therapeutic value in suspension.

As to the case of doubtful nature which we have included among those who benefited by suspension, he came to hospital in August, complaining of pains in his legs, and after examination his knee-jerks were noted as absent. His pupils reacted normally, he had no inco-ordination, but slight starting of the tendons as he stood, no bladder trouble, no anæsthesia. Unfortunately his knee-jerks were not examined during the time he was suspended, but on examining him after his complete course—he was an out-patient—his knee-jerks were found active. His pains were less. What the nature of the case was it is not easy to say, but there is no evidence in favour of tabes except the absent knee-jerk, and as this was only observed on one occasion and by one observer, it can hardly be accepted as convincing.

Of the other cases, as we have said, five became worse while undergoing treatment, viz., three of tabes, one of spastic, and one of ataxic paraplegia. Of the three, one was a case pursuing a rapid downward course, and this course was in no way checked by suspension; a second was an extremely ataxic patient just able to walk with strong support, with bad cystitis and incontinence, and subject to pains and gastric crises. His treatment suffered interruption by a severe attack of gastric and vesical pain, accompanied with vomiting, and after it was resumed and completed, his cystitis and incontinence were still bad, although his bladder had been washed out daily; he was still subject to attacks of pain and his power of walking was lessened. The other case of tabes which became worse was an out-patient. After nine suspensions he remained away for a week on account of severe lumbar pain, and on returning his walking was manifestly worse. He had one more suspension, and then had an attack of erysipelas of the face, which did not allow resumption of the treatment.

The case of spastic paraplegia which became worse, was a young man with the disease affecting chiefly the left leg, and after he had had thirty suspensions this leg was considerably more rigid than it had been. The patient with ataxic paraplegia suffered from nausea or vomiting at or after each suspension, and although he had only had four he was weaker

and more shaky than he had been before the treatment was commenced.

Of the cases which remained *in statu quo*, most had ataxy, a few had occasional attacks of pains, some had cystitis and incontinence, one or two had gastric crises. Those symptoms underwent no change. Six had optic atrophy as a complication, of whom two were completely blind. No improvement was experienced in those cases, and in the cases where vision was less affected, careful testing could reveal no improvement, although one patient expressed the belief that he could see better. Dr. James Anderson has had this patient under observation since, and he informs us that there is now a distinctly greater impairment of vision.

It is evident that our results are not to be compared with those obtained by the majority of observers. While there are a few isolated observers, such as Bernhardt, Erb, Dujardin-Beaumetz, and Saundby, whose results are no less discouraging than ours, the great majority seem to have been far more fortunate. In the preceding lists there are in all 255 cases of tabes, of whom

171 improved,
77 did not improve,
7 became worse.

Our list included 32 cases of tabes, of whom
6 improved,
23 did not improve,
3 became worse.

To put it differently:—the percentage of improvement in other cases is 67 per cent., while in ours it is 18·7 per cent.; the percentage without improvement in those is 30·2 per cent., while in ours it is 71·9 per cent.; while in those cases 2·8 per cent. became worse, in our cases 9·4 per cent.

As perhaps explaining in part this great divergence, it must be remembered that while with any new treatment such as this, everyone who gets good results will at once be inclined to publish them, those who are not so fortunate and who are perhaps less sanguine, will not have the same eagerness to announce their unfavourable results. Two instances

in which this has been the case have come under our own notice, and we have no reason to suppose that our experience is unique. Then as to any great innovation in therapeutics we may be sure that its first year is its best. The enthusiasm for it dies down later, and the results are no longer seen in the rosy light which accompanies the dawn of any new treatment. We have only to look back a few years for an example of this, to the introduction of nerve-stretching for the relief of this same disease. The feeling with which a perusal of the results of that treatment in its first year will inspire anyone now is one of surprise, that any treatment offering such marvellous results should have fallen so soon into disrepute and disuse.

We have not yet cases of disease other than tabes in sufficient number to allow us to draw any definite conclusions as to the effect of suspension. Some cases of paralysis agitans have improved a little while being suspended, the majority have not. The results in spastic paraplegia are varied, while in disseminated sclerosis the general consensus of opinion is against any good effect of suspension. That the sexual apparatus is affected in some way in a certain number of cases is evident. That actual increase in power is caused is, we should think, extremely doubtful. Over excitation is a much more likely result, and whether this is to be regarded as an improvement or the reverse will depend on the nature of the case.

With the experience which we have had of suspension in cases of tabes it will readily be understood that we are not inclined to give the treatment much credit for relief, and far less for cure of any of the serious symptoms. In endeavouring to gauge the value of any therapeutic agent in this disease the peculiar and varying character of its manifestations must be borne in mind. One of our greatest authorities on nervous disease states the percentage of cases shewing no progressive tendency so high as 50 per cent., and every one knows that in many cases actual and marked improvement takes place both under treatment and independently of it. There was lately in this hospital, under Dr. Buzzard's care, a patient with tabes—a patient who, eight years ago, was

unable to walk from extreme ataxy and weakness, but who, during the last three or four years had been steadily improving and who is now (although the disease is well marked) able to walk about without even a stick, and to carry out efficiently the duties of military librarian in one of our garrison towns.

Reference has already been made to Dr. Churton's case, which improved distinctly while waiting for the suspension apparatus to be mended. Had this case been suspended, the improvement would naturally have been ascribed to the suspension, and such cases show how extremely careful one must be about *post* and *propter*. That ataxy varies at different times is well known. The familiar case of a patient who immediately after a severe attack of lightning pains has a marked increase of inco-ordination, illustrates this. Suppose such a patient towards the conclusion of the attack of pains subjected to suspension treatment, there is every chance that the cessation of the pains and the subsequent improvement in gait will be credited to the treatment, although numerous cases make it evident that improvement would follow even if nothing were done.

Lightning pains themselves come and go in the most unexpected way. Between one paroxysm and another there may be an interval of a few days; between that and the next there may be as many months; or they may vanish and never return. The very cases in which they have persisted for some time are those most likely soon to recover from them, for after all, their duration is limited, and this must be borne in mind in estimating the value of suspension, which by many is claimed as so efficacious in those pains. In none of our cases were pains a severe symptom before treatment. They did occur occasionally and they did not seem to be affected by suspension.

Gastric crises, too, relief from which, though to a much less extent than from pains, is claimed as a result of suspension, are just as variable and capricious as the pains. They, too, may vanish and never return, or they may return in a day or a year. That they had gone permanently could only be claimed after prolonged observation, and even then without a very large number of cases and without much

more uniform results than have as yet been obtained, their disappearance could not be ascribed to the treatment.

In none of our cases were gastric crises a very severe symptom. They had recently been present in very few, but where this was the case they continued to obtrude themselves at times. In one case a severe attack of gastric and vesical pain, accompanied by sickness and vomiting, necessitated an interruption of the treatment for a fortnight. We do not think that we should be justified in ascribing this crisis to the treatment, any more than we think others justified who do not hesitate to ascribe the absence of such symptoms to suspension.

The bladder troubles also in tabes are variable, and depend so much on mental conditions and on general physical conditions, both in and outside the body, that the greatest possible care is necessary in drawing conclusions as to the effect of treatment on them. In the majority of the cases reported, improvement in these symptoms is noted, but as in at least three cases—one of Michell Clarke's, one of Eulenburg and Mendel's, and one of Bernhardt's—there was greater trouble in this respect, while in another of Michell Clarke's, a tendency to incontinence was replaced by a tendency to retention, we must refuse to credit suspension with the improvement in one set of cases unless it is also made responsible for what happened in the other series. In our cases we have observed no change. Where cystitis was present the incontinence remained, and that too, although the bladders were daily washed out. In the other cases where urinary trouble was present, this consisted in slight difficulty in commencing the act, or in difficulty in resisting the call. In none of the cases was the symptom a troublesome one, and it remained unchanged. In the recorded cases in which incontinence was a symptom it is not stated whether cystitis was present and whether any local treatment was adopted. If the latter was the case, then it will become a question how much of the improvement is due to the local treatment. So far as we are aware, the only case in which a cure of cystitis by suspension alone is claimed, is one referred to at a recent meeting of the Medical Society of

London. Such a result is, of course, extremely unlikely, to say the least of it, and we should certainly hesitate to accept it without further details.

As to the increase or restoration of sexual power which is claimed as the result of this treatment, we have not observed it in any of our cases. A greater tendency to erection has manifested itself in at least one instance, but where sexual power was gone it has not been restored. Such a result of suspension, however, is claimed by some, while on the other hand one observer claims to have stopped the occurrence of troublesome erections by this treatment. In another case—one of Bernhardt's—there occurred during treatment an excessive tendency to erection with seminal emissions, so frequent and so distressing that the patient begged that suspension might be discontinued.

Passing by the question as to the advisability of restoring sexual appetite or function in tabetics, especially in cases where the disease is stationary, we can only say that this restoration after suspension treatment is by no means constant, and that instead of it the patient may experience a kind of false restoration, and may find himself in a condition much more distressing than his original state.

So much for the varying symptoms of tabes. But there are signs which do not vary, or at least not to the same extent, viz., ocular paralysis and impaired vision, the Argyll-Robertson pupil and the loss of knee-jerk. Of these there is perhaps the greatest chance of variation in ocular paralysis, especially in paralysis of the Levator palpebræ. In one case it is claimed that ptosis had disappeared, but as it frequently does so under the most diverse forms of treatment, its disappearance under suspension is not of much significance. In no case is it claimed that diplopia disappeared, but M. Darier claims improvement in vision in four cases with optic atrophy. Although a good many such cases are recorded, this observer is the only one who claims improvement. Among our cases are six with optic atrophy, and no improvement resulted in any, although one thought his sight better. Testing did not confirm this impression.

But more changeless than these are the loss of the knee-

jerk and, when it is present, the Argyll-Robertson pupil. In one case where the latter was present it is claimed that after suspension the pupil reacted sluggishly to light. Everyone knows how very difficult it is sometimes to be sure whether the reaction to light is present or absent. To illustrate this, we may mention a case which was seen about a year ago by a physician of eminence and in whom it was noted that the pupils did not react to light. Three months later the pupils reacted to light, and they do so still, and I think the physician himself would be the first to acknowledge that this was due, not to treatment, but to an error of observation in the first instance.

As to the return of the knee-jerk, the facts to establish such a change must be incontrovertible. The violent jerk, after five or six taps, claimed by Marina as a return of knee-jerk is to be regarded with the greatest suspicion. A very inactive knee-jerk cannot be elicited in that way, and we should think it is much more likely to have been a voluntary phenomenon. As to Dr. Althaus's case, in which the knee-jerk returned, as the case is only somewhat cursorily described in a letter to the *British Medical Journal*, we shall require further details before we can decide as to the real nature of the case. We think it may be laid down as an axiom that in a doubtful case the evidence of a solitary observer—no matter who he may be—as to the presence or absence of knee-jerk, should always be re-inforced by that of at least one other.

Such are the considerations which it seems to us necessary to urge before we arrive at a true appreciation of the value of any treatment of tabes. We have not referred to the mental effect likely to be produced by an impressive treatment upon patients who as a rule have tried many remedies, unfortunately in most cases with little success, and who are only too willing to clutch at even the shadow of a new means of cure. That such mental effect is considerable in some cases is certain, and how far it will account for that feeling of well-being, and of improvement in gait and balancing power, which wants the confirmation of any objective sign, it is not easy to say, but that some of the improvement is of this nature, I think, may be assumed. That it will

vary in different cases is evident, and we may possibly be able to detect national, as well as individual differences. It is not easy to decide how far such a consideration is to be allowed to influence a medical man in the treatment of a patient. That it should have some influence we think all will be prepared to admit, but to say as has been said[1] that "the patient is to be the final judge of treatment" is more than we should be prepared to admit. Whether this means that the patient is to have the last—and presumably the decisive voice—in deciding on the nature of treatment, or whether it means that the patient is to draw conclusions as to the value and efficacy of treatment in his own particular case—conclusions which are to be accepted by medical men—in neither case, we think, can the dictum be accepted. If it be accepted, the physician will certainly be relieved of considerable responsibility, but we think it will then be difficult for him to justify his existence at all. It seems to us that it rests with the medical profession to decide on the value of any treatment. It is for that body to examine and sift the evidence for and against; it is for the individual members to collect it, and lay it before that tribunal. We trust that our contribution may not be without its use in enabling an estimate to be formed of the value of the treatment by suspension.

[1] *Lancet*, Jan. 4th, 1890.

PRELIMINARY NOTE ON CERTAIN MORBID PRODUCTS FOUND IN THE BRAINS OF PATIENTS DYING AFTER SEVERE HEAD INJURIES.[1]

BY ALEXANDER MILES, M.B., L.R.C.S., EDINBURGH.

WHILE engaged recently in investigating the microscopic appearances in a number of brains of patients who had died at varying periods, after receiving severe head injuries, I found in several cases large numbers of peculiar bodies, which appear to me to bear some relation to the so-called Colloid bodies described by various writers.

These bodies were found in the brain of a youth who died fourteen hours after sustaining severe injuries to his head by falling off the Forth Bridge; but as they were more characteristic in the case of a mason who survived his accident for two-hundred and fifty-six hours (256), I shall describe them from sections of his brain.

The bodies referred to vary in size from 7ω to 50ω; the average being about 18ω. In shape they are more or less rounded or ovoid; are homogeneous, with no evidence of a nucleus, or other cellular contents, and except when very deeply stained are perfectly translucent. In some cases they exhibit a regular outline, the periphery being somewhat more deeply stained than the rest of the cell, while in others the margin is sinuous giving it the appearance of having shrivelled up slightly.

They take up the hæmatoxylin stain very readily, and stain black with osmic acid, but are unaffected by aniline-blue-black, and picro-carmine. The smaller examples stain faintly and uniformly, while the larger ones vary considerably in the degree to which they react to the dye. It would appear

[1] From investigations conducted in the Research Laboratory of the Royal College of Physicians, Edinburgh.

that these latter are formed by the coalescence of several of the former, as is shewn by their irregular outline at first, and later by their tense rounded appearance.

They are not confined to any one region of the brain, but are found in greater or less numbers alike in the cortex, basal ganglia, pons, medulla, and cord. To describe them as they occur in the cerebral hemisphere

They are distributed:—

(1) All through the white matter.

(2) In the most superficial area of the grey matter.

(3) In the "lymphatic system" of the brain.

(1) *In the white matter* they are exceedingly abundant, being thickly studded all through the section. Here they are fairly uniform in size and take up the stain well. They are largest, however, and most numerous in the immediate vicinity of punctiform hæmorrhages into the brain substance, where they not only invade the torn strands of nerve fibres, but also pass among the extravasated blood corpuscles.

(2) *In the grey matter* they are only to be detected in the most superficial layer, but there they are very plentiful. While they are less darkly stained and smaller than their fellows in the white matter, they shew a greater tendency to coalesce with one another, especially on the free surface of the brain, the largest being found in the epicerebral space.

(3) *In the "Lymphatic system" of the brain* they are chiefly found in one of two places, viz.:—

(*a*) In the meshes of the pia mater—the subarachnoid space.

(*b*) Along the course of the larger intra-cerebral blood vessels.

(*a*) It may be of importance from the point of view of their origin to observe that when these bodies are found in the *sub-arachnoid space* it is always in close proximity to a laceration of the brain substance, which makes it probable that they are then derived from the superficial layer of the grey matter. It is in this situation that they attain their greatest size, possibly because they have more room to expand here than elsewhere, or it may be that they swell up by imbibition of cerebro-spinal fluid.

(*b*) When in relation to *intra-cortical blood vessels* these bodies are found in the perivascular lymph space of His, between the hyaline membrane, or adventitia, and the brain tissue. They also are present for a short distance into the brain substance, and sometimes find their way into the space of Robin, between the tunica adventitia and the tunica muscularis, whether by natural or artificial openings is difficult to determine. The vessels round which they are seen are most evident in the superficial layers of grey matter, next in the region where the grey and white matters join—a very vascular area—and to a less degree as we pass deeper. In all respects they resemble those described in the sub-arachnoid space, save that they are somewhat smaller and more compressed laterally.

Of the exact significance of these bodies I am not at present prepared to speak definitely, but their general appearance, distribution, and reaction to staining agents, seem to point to their being in some way related to the myeline of the nerve fibres. Probably the main mportance of the observation lies in the demonstration that these bodies may be produced very rapidly.

Clinical Cases.

COMPLETE PARALYSIS OF RIGHT THIRD NERVE IN A PATIENT AFFECTED WITH LEAD PALSY —RECOVERY UNDER THE DIRECT APPLICATION OF GALVANIC CURRENTS.

BY THOMAS BUZZARD, M.D.

Physician to the National Hospital for the Paralysed and Epileptic.

H. J., æt. 38, was admitted under my care into the National Hospital for the Paralysed and the Epileptic on February 4th, 1889, affected with complete paralysis of the right third nerve.

It appears, from notes taken by Dr. Taylor, Resident Medical Officer, that until five years ago his occupation had been that of a venetian blind maker, and that his health up to that time had been good. He then began to suffer from symptoms of lead colic, and a month or two later from "numbness," first in the little fingers and then in the other fingers of both hands. His hands became weak, so that he could not grasp anything strongly. He had also, he says, great difficulty in straightening his arms, and his hands hung from the wrists. His hands improved under treatment after about six months, but then he began to have attacks of headache, from which he has suffered from time to time ever since. The headache is described as affecting the right side of the head and forehead and being worse at night.

About a month after this headache commenced he had a fit, of which he knows nothing except that he bit his tongue severely. He had no return till eighteen months before admission into the hospital, when he was convulsed but did not bite his tongue. He had five or six of these apparently severe general fits, and then others of different and unilateral character. In these last he did not lose consciousness; the right side of the face was drawn up and the right arm and leg "worked up and down." Before the fit he always had trembling in the right arm and leg. In the attack he could not speak, though he understood what was said to him. He would have such attacks about every other day for eight or nine months, but none had occurred for four or five months before admission. He became an inmate of several hospitals in turn.

Four or five months before he came under my care he suffered from some loss of feeling on the right side of the head, but his

account of this is very indefinite. On January 31st, 1889, he woke up and found he could not open the right eye. There had not been any pain in the head just before.

The patient is married. His wife has twice miscarried, and he has three living children; the eldest delicate, very dull, and it would seem mentally affected; the others healthy. One died of some unknown cause. He denies any veneral infection.

On admission he was described as a pale, anæmic looking man, complaining of pains in the arms, weakness in the hands and legs. There was ptosis of the right eyelid, with abolished movement of upper, lower, and internal rectus muscles. The pupil was dilated, and did not react.

In the left eye Mr. Gunn reported that the edges of the disc were blurred, and there was a haziness over it.

A well-marked blue line was found on the gums.

In the arms there was but feeble power of extending the wrist joint, and the extensor communis digitorum and proprius pollicis failed to react to faradism. Omitting detail, the condition of the forearms was typically characteristic of lead paralysis.

The treatment adopted was at first indifferent, during which his symptoms remained unchanged. On February 13th he was ordered twenty grains, and on the 26th, thirty grains of iodide of potassium, to be taken three times a day. Under this the patient expressed himself as feeling generally better, and his arms stronger, but no difference shewed itself in the condition of the right eye.

On the 20th March, at his own request, he became an out-patient.

At this time he had been taking from sixty to ninety grains of iodide daily for thirty-five days, and there was no power whatever in lifting the lid or in moving either of the affected ocular muscles.

The constant current slowly interrupted was now applied in the following manner. A well-wetted plate rheophore was applied to patient's nape of neck, and connected with one pole of a Leclanché battery. The operator took the other rheophore well-wetted in his left hand, grasping the metallic portion in his palm, and applied the current to the patient's eye with the point of the index finger of his right hand. This was covered with one thickness of muslin, well wetted. The conjunctiva had been first rendered insensitive with a 2 per cent. solution of hydrochlorate of cocaine.

The strength of current which was employed marked from 1·5 to 2 m.a. on the galvanometer, and the alternate application

and lifting of the finger, making and breaking the circuit, gave rise to the feeling of an electric shock in the terminal joint of the operator's finger. The strength of the current was such as just to give rise to this sensation, and it was previously tested upon the patient's cheek.

Applications were then made in turn to the upper lid and the conjunctiva as near as could be contrived to the insertion of the internal, upper, and lower recti. The sitting occupied about two or three minutes.

A week later, on March 26th, when he was again seen, it was found that he could separate the lids by an eighth of an inch. The application was renewed, and on April 2nd it is noted that the "lids are now constantly apart. The pupils are equal. The right acts during convergence, but not for light."

On the 9th April Dr. Taylor's note runs thus: "The lower edge of the upper lid is now on a level with the upper border of the pupil. There is slight movement of the superior rectus—the inferior and internal recti remain as before. The pupils are now equal in size." He now felt his gait awkward, owing to erroneous projection, which he had hitherto been spared in consequence of the ptosis of the lid. There was marked diplopia. The galvanic applications were continued twice a week.

On April 16th it is remarked that the eye does not pass inwards beyond the vertical meridian of the palpebral aperture. On the 23rd the resident officer notes: "The right pupil is now slightly smaller than the left. Both react normally in accommodation. The right eye is as widely open as the left. The upward movement of the right globe is not quite so good as that of the left, and the downward is still more decidedly impaired, so also is the internal movement, but the eye now passes inwards beyond the vertical meridian of the aperture."

By May 15th the movement of the internal rectus was much improved, as likewise was that of the inferior, whilst the superior rectus appeared to act almost normally.

The patient was somewhat irregular in his attendance, so that the electrical applications were not made more than once a week.

In June the only defect remaining was an absence of reaction to light in the pupil of the right eye. All the external ocular muscles acted normally.

This case is marked by several features of interest. Although the gums presented a well-defined blue line, the patient had not been exposed to the action of lead in his

occupation for five years before he came under observation, and no source of more recent lead-poisoning could be discovered. There was a history of a few fits, apparently of epileptic character, the first about five years, and then none till eighteen months before admission, when he had a return of some half-dozen of the same character. These were followed by very numerous epileptiform attacks, apparently very different from the previous seizures, and pointing to some cortical lesion. The presence of optic neuritis lent confirmatory evidence in the same direction. The history of facial anæsthesia preceding the paralysis of the right third nerve is suggestive of some gross lesion in the basis cranii, affecting in succession the fifth and third nerves of that side.

What was the cause of these symptoms? It is natural, in the presence of a characteristic blue line, and of the typical features of lead palsy to ascribe the epileptic and and epileptiform seizures, as well as the paralysis of the right motor oculi nerve, to the action of lead. But five years had past since the patient was, so far as is known, exposed to the action of that poison.

Moreover, although convulsive seizures are not unfrequently met with, I have never previously seen or met with any record of a case in which complete paralysis of a third nerve could be reasonably referred to the action of lead. It is true that Professor Westphal has recently published some cases of plumbic encephalopathy, in one of which paresis of one abducens oculi muscle was noted.

If the intracranial symptoms described were not due to lead it is, I suppose, most reasonable to refer them to syphilis, and to suppose that they were dependent upon gummatous deposit on the cortex cerebri, and also in the basis cranii. Such a concurrence of lesions is, of course, common enough in syphilis. To this view, however, there are two objections —one, not strong, that the patient denied any veneral disorder; the other, in my opinion a more weighty one, that although he was put under active specific treatment within fourteen days of the attack of ocular paralysis, no impression whatever was made upon this condition by large doses of iodide administered during thirty-five days. I could not

venture to say that this circumstance contra-indicated the syphilitic nature of the lesion, but I think that it throws some doubt upon it. On the other hand, the preceding lesion in the district of the fifth nerve, as well as the completeness of the eventual recovery, speak much more strongly in favour of syphilis than of lead, and appear to point pathologically to neuritis rather than to nuclear lesion.

The mode of application of the galvanic current in this case being unusual, and its results happy, I am tempted to give a few particulars of another recent instance in which the patient was favourably influenced by like treatment, though not to the same extent as in the case just related.

A married lady of about thirty-five years of age was seen by me in consultation on October 26th, 1886, suffering from complete paralysis of the left third nerve. For five weeks she had suffered from very violent pain over the left brow night and day, and for a week past this had been accompanied by severe and frequent sickness. Her condition shewed extreme suffering, and for several nights she had had no sleep at all.

She was ordered ten grains of iodide every four hours. On October 28th the pain was perhaps a little less, but still most severe. The dose was increased to fifteen grains. On the 29th she was still in great suffering. The iodide was continued, and in addition she was ordered calomel gr. $\frac{1}{4}$, with opium gr. $\frac{1}{8}$ every three hours. She got relief after the first pill, and when I saw her on November 1st she was able to eat, and appeared to be greatly improved so far as the pain was concerned. The dose of iodide was now increased to twenty grains every four hours. On the afternoon of that day the pain returned with great severity, accompanied by retching and vomiting. Morphia was now given by injection.

On November 4th the pain quieted down, and was never afterwards severe. The condition of the eye, as regards the paralysis remained unchanged. There was at no time any conjunctival hyperæmia or tenderness of the globe. The ophthalmoscope shewed no change.

The mercurial treatment was continued to salivation, and the iodide was taken three times a day, until the end of November, without however producing the slightest perceptible effect upon the paralysis of eye muscles. Early in December she was seen by

Mr. Hutchinson, who was disposed to give an unfavourable prognosis. She continued during the month in the same state.

On January 12th, 1887, when all specific treatment had been suspended for some weeks I decided to apply electric currents. The left third nerve had then been completely paralysed for more than three months, and there was not the slightest sign of any disposition to return of power. On January 12th I applied a constant current slowly interrupted in the manner just described to patient's upper lid. After a few minutes' application there appeared to be a very slight return of power in the levator palpebræ. A few days later this was a little more marked. The application was now made to the various affected muscles, and there was immediate, though slight, improvement also in these, especially in the internal rectus. After four applications, the power of the levator palpebræ appeared to be perfectly restored, the left eye being opened as widely as the right. There was diplopia on looking to her right. At the same time on each occasion the power of the internal rectus was found to be increased, and after about eight or ten applications this muscle had entirely recovered.

The patient, it should be said, being professionally occupied, her attendance was very irregular, and sometimes at long intervals. In October, 1887, after about twelve applications I made the following note: "The left eyeball now rolls upwards better—the pupil is larger than that of the right side. It does not contract to light, but reacts slightly during the effort at accommodation. There is no action in the inferior rectus. The internal rectus acts perfectly."

In January, 1888, I found that when she looked to her *left* there was some diplopia, the images being described as about four inches apart. Examination now shewed that she failed to bring the cornea to the *outer* canthus.

In November last this inability had slightly increased so that the cornea, when she looked to the left, remained at about $\frac{1}{4}$ inch. from the outer canthus. The diplopia necessarily continued. At that time there was some, but imperfect movement of the superior rectus and still less in the inferior rectus, though this was not absent. The internal rectus and levator palpebræ remained in perfect order. The condition has since not materially changed.

I would suggest the possibility that the apparent weakening of the external rectus may be due to some secondary contraction in the internal rectus muscle. One often sees in cases of peripheral paralysis of the portio dura that after recovery the contraction of previously paralysed muscles by over pulling the face gives rise to

apparent paralysis of the sound side, and it occurs to me that an analogous condition may possibly sometimes obtain as regards the internal and external rectus muscle of the eye.

We are justified, I think, in feeling that the striking and immediate effects which followed the application of the galvanic current in these cases is sufficient to indicate a relation of cause and effect, and not a mere association of coincidence. I have seen others of a similar kind, but my notes of them are not complete enough for publication.

As is well known the galvanic current is usually employed in such cases indirectly through the closed lids. It is hard to conceive that a current sufficient to influence the external ocular muscles can be carried to the requisite depth, through the resistance of the lids, without employing a strength which would be quite intolerable to the patient, and I have little doubt that the supposed galvanisation of these muscles through the lids is practically inert. The method of direct application described above was introduced by me in a paper published in the *Lancet* in 1875. It has been favourably noticed by Erb, but otherwise does not appear to have attracted attention. At the meeting of the Ophthalmological Society on July 5th, 1889, I demonstrated the mode of application.

PARALYSIS ESPECIALLY OF ONE DELTOID MUSCLE IN A PATIENT SUFFERING FROM LEAD POISONING; PRESERVED ELECTRICAL REACTION OF THE MUSCLES.

BY THOMAS BUZZARD, M.D., F.R.C.P.

Physician to the National Hospital for the Paralysed and the Epileptic.

E. G., æt. 45, a house painter, was admitted into the National Hospital for the Paralysed and the Epileptic on October 8th, 1889, when the following notes were taken by Dr. Taylor, House Physician :—

Patient was in his usual health until three weeks ago, when he was suddenly seized with severe abdominal pain and constipation which yielded after three or four days to treatment. He then noticed that his hands and his shoulders, especially the left, were weak. For several weeks before the abdominal attack he had observed some weakness in both thumbs—a difficulty in abducting them, and a cramp-like pain in both little fingers. He had also noticed for some time past shakings in both hands, and this at times during the past summer had been so bad that he had been forced to stop work for a while. He had been obliged, for example, to employ both hands in taking a cup of tea. There had been, besides, pain in the left knee and both shoulders, and also in the left elbow. He had had no headache.

Five weeks before admission he had accidentally cut the back of his right hand with glass.

The patient had enjoyed good health, with the exception of occasional attacks of colic. In his work as a house-painter he had to do much "flatting." He is married, and has six children all healthy, and his wife had had no miscarriages.

On admission, patient is a healthy-looking man, complaining of weakness in the hands and arms, especially on the left side, and pains in the knees. Nothing wrong is to be noted about the face and head. There is a well-marked blue line on both upper and lower gums. All movements of the fingers are carried out fairly well, but with tremor, and there is a tendency to flexion of the fingers at the metacarpo-phalangeal joints.

Right Arm.—The thumb is kept in a position of flexion at all the joints. Flexion, adduction, and opposition are carried out

fairly well, but with tremor; extension and abduction cannot be performed.

In the wrist the movements of flexion and extension as also ulnar and radial flexion and extension are carried out with fair power. Extension at the wrist is certainly the weakest movement. The movements of the elbow and shoulder-joints are good.

Left Arm.—There is here also a tendency to flexion at the metacarpo-phalangeal joints. The fingers however can be extended, and flexed, and separated from each other. The condition of the thumb is similar to that on the right side.

At the wrist all movements are carried out, but extension is executed feebly, and with tremor.

Left Shoulder.—The arm cannot be raised any distance from the side, nor can the extended arm be retained at a right angle to the trunk, or raised above the head. In attempts to do this much fibrillary contraction is noticed in the deltoid muscle.

Tremor is present when attempt is made to do anything, and this is equally well-marked in each hand. Fibrillary twitching is apparent in the forearms, especially on the extensor surface. There is no incordination of movement. The elbow and wrist jerks are active and equal.

In the lower extremities all movements are carried out well, and there is no marked tremor. The knee-jerks are active and equal; there is no ankle-clonus; the plantar reflex is active and equal. Nothing wrong is to be noted in the thoracic and abdominal cavities. The urine is acid, sp. gr. 1020; no albumen; no sugar.

Examined electrically it is found that all muscles of both arms and hands—including the deltoids—react equally to a faradic current of a strength not greater, as far as can be judged, than would be necessary to cause contraction in healthy muscle. Reaction to the galvanic current is normal.

During patient's stay in hospital some little atrophy of the left deltoid became observable, but the electrical reaction continued normal.

Primary paralysis of a deltoid muscle in a case of lead poisoning, although rare, is by no means unexampled. Duchenne (de Boulogne), in his "Electrisation localisée," remarks: "The deltoid muscle is sometimes paralysed primarily," and refers to a case which he saw in hospital. The patient was affected with paralysis of the right deltoid consecutive (as in this patient) to several attacks of lead

colic. In Duchenne's patient, however, the middle fibres of the muscle had completely lost their faradic excitability, which still remained, but in a very feeble degree, in the anterior and posterior third. In the case which I bring forward the most remarkable feature is the preservation of electric excitability, of which I do not remember to have seen another example in a case of lead paralysis, where the loss of power was as complete as it was in this case. Indeed, the converse is what is to be expected. In an early number of this Journal[1] I pointed out that some muscles supposed by a patient suffering from lead paralysis to be healthy and unaffected, showed great diminution of faradic excitability, a point which had also been dwelt upon by Erb and Bernhardt before me.

When this case first came under my care a serious question arose in my mind (from observation of the electrical condition) as to the bona fides of the patient. But I soon came to a definite opinion that simulation was out of the question. The weakness of extensors shown by the tendency to flexion of the fingers and wrist, the tremor, the very distinct blue line, coupled with the history of repeated colic, bore powerful evidence as to the genuineness of the case. But most striking of all, perhaps, was the action of the man when asked to lift his elbow away from his side. Instead of alleging his incapability of doing so, he would make strenuous efforts to bring up his elbow, by employing the pectoralis major muscle in a manner unmistakeably characteristic of a case of deltoid paralysis.

Moreover, although on his admission no atrophy of the left deltoid was to be found, a perceptible thinning of its fibres occurred during his stay in hospital. Lastly, the man always endeavoured to make the best of his condition, and was with difficulty persuaded to remain in hospital, as he thought he could manage to do a little work.

When this patient was exhibited before the Neurological Society of London, at its meeting on November 21st, the preserved electrical reaction of the deltoid muscle was demonstrated, in my unavoidable absence, by Dr. Taylor.

[1] *Brain*, Vol. i., page 121.

ILLUSTRATIONS TO DR. SHARKEY'S CASE OF DIPHTHERIA WITH ABSENCE OF KNEE JERKS.

A CASE OF DIPHTHERIA, WITH ABSENCE OF KNEE JERKS.

BY SEYMOUR J. SHARKEY, M.D., F.R.C.P.

Wm. C., æt. 4, was admitted into St. Thomas's Hospital, under my care on August 18th, 1888, and died on August 23rd, 1888. By some mistake I never saw the child during life.

He had been strong and healthy all his life (except for measles at eighteen months) until two months before admission, when his mother and her four children, the patient among the number, had sore throats, foul breath, and pain in the ears. No doctor was called in. After the throat got well the patient spoke with a nasal twang, fluids regurgitated through the nose, the voice got weak, so that he could barely make himself understood, and he became hardly able to walk.

On admission temperature was 98.6 F.; pulse 120, regular; tongue moist and coated with white fur; appetite said to be good, and bowels regular.

Body well nourished, no wasting of muscles observable. The patient used his arms and legs naturally, and could walk, but somewhat feebly.

Speech was indistinct and nasal in quality and difficult to understand. In drinking he choked a little, but there had been no regurgitation since admission. He made a snoring noise in respiration, and coughed a little. Soft palate and uvula did not move well. Throat not swollen or inflamed, but contained much mucus, chest moved well; no obstruction to the entry of air, physical signs normal. Heart normal. Knee-jerks absent. Urine 1025; no albumen or sugar. Eyes natural in every way.

During the rest of his life the temperature was often slightly elevated, and owing to difficulty in swallowing he had to be fed by a nasal tube. A trace of albumen appeared in the urine, and he got rapidly feeble.

On August 22nd he had two bad attacks of dyspnœa, attended with soft stridor. The diaphragm and inter-costal musles were acting fairly well, and there was no evidence of pulmonary disease. As these attacks made feeding very difficult, and as the breathing was evidently obstructed in some way tracheotomy

was performed. On the day following it he had two or three attacks of dyspnœa, not so severe as before, and blueness and pallor came on suddenly. The pulse was fairly good and temperature normal. He died suddenly on August 23rd, apparently from stoppage of respiration.

Post-mortem Examination.—Body pale and thin, but well formed; heart, liver, lungs and spleen were all perfectly healthy in appearance. Lungs shewed here and there slight collapse, but were otherwise normal. Brain was generally hyperæmic, otherwise natural. Spinal cord looked healthy throughout.

The tonsils were somewhat enlarged, but free from membrane or any signs of acute inflammation. The soft palate and uvula were thick, almost œdematous in appearance. Larynx normal.

Thinking this was a favourable case for investigating the cause of the absence of the knee jerk in diphtheria, I hardened and had cut for me the spinal cord, the anterior crural nerve, and the quadriceps extensor muscle. The nerve and muscle proved to be perfectly healthy, but the spinal cord, though in the main normal, presented a very peculiar pathological alteration. The seat of it was in the large motor cells of the anterior columns; in them the change was very marked, but I was unable to make out any such alteration in the smaller cells of the posterior cornu. There were no evidences of inflammation to be found, and the other constituents of the cord were natural. The alteration in the multipolar cells is well reproduced in the figure, and consisted of a number of very large granules, completely filling the cells, and taking the logwood stain very deeply.

So numerous were they that they often completely obscured the nucleus. Their was a peculiar regularity about their arrangement, which is well seen in the figure, and the processes of the cells were often beset with them. In the latter situation they were long and appeared parallel with the direction of the process. The appearance was no ordinary granular condition, seen sometimes even in normal cords, but something quite unusual. Mr. Sherrington to whom I showed the sections said he had never seen such a condition before. On looking through a variety of preparations the only cases in which I found something of a similar kind were cases of myelitis.

The pneumogastric nerve I also examined microscopically and found it quite healthy. In several other cases of diphtheria which have come under my notice I have examined the paralysed nerves and muscles and found them healthy.

In the present case the alterations in function produced by

the disease were widespread and severe as was proved by the presence of the nasal twang and regurgitation of fluids through the nose: by the weakness of his voice, and his difficulty in walking: by the absence of knee jerks, difficulty in swallowing, the trace of albumen in the urine, and finally by his death from stoppage of respiration. I am lead to believe that the paralytic phenomena, and the absence of knee jerks were due in all probability to a change in the motor cells of the central nervous system, partly because the examination of nerves and muscles in this and in other cases proved negative, and partly because of the alteration found in the motor cells of the spinal cord.

The question naturally arises what gave rise to these changes. At present we are not in a position to make any definite assertion on this point. But if, as is probable, diphtheria is produced by micro-organisms, we can readily suppose that the vital changes which these organisms undergo in their growth upon the surfaces of the body and possibly in its interior produce certain poisons which being absorbed act specially as nerve poisons: and that the changes observed in the motor cells of the cord in the present case were the results of the action of such a poison. This is more likely than that the micro-organisms produce these results by their actual presence. For one cannot demonstrate their presence by the ordinary methods, and the paralytic phenomena often present themselves after all evidence of the original disease has disappeared, if we may judge by the absence of fever and of local inflammation. But the change in nerve cells produced by such a poison might well take considerable time to be recovered from. It does not seem to me that it would be fatal to this theory if in future examinations cases in which the knee jerk may have been absent, and the patient may have died from pulmonary or other troubles, no changes should be found in the cells of the spinal cord. For the poison might act in a lesser degree without producing serious disorganisation of nerve cells: and the knee jerks appear to be a singularly delicate test of the complete healthiness of the reflex arc in connection with them, and are abolished by slight causes.

If, however, changes should be absent in cases which have run so severe a course as the present, and which have been marked by serious and widespread paralytic phenomena, then the alterations found by me in the motor cells in this case will have to find some other explanation.

Critical Digests.

CHRONIC HYDROCEPHALUS.

BY M. ARMAND RUFFER, M.A., M.D.

(*Continued from page* 144.)

II.

Owing to the varying position of the lesions, it is impossible to give a clinical account applying to all cases of hydrocephalus.

The brain being distended by fluid, in one part or as a whole, it follows that the symptoms vary not only according to the amount of destruction of brain-tissue, but also according to the part of the brain which is the seat of disease. It will be better therefore to divide the subject into the same two groups, as in the part referring to the pathological anatomy of the disease, namely:—

1. Porocephalus, *i.e.*, Localised or total destruction of the brain tissue by a collection of fluid.

2. Hydrocephalus, *i.e.*, Distension of the brain by fluid without loss of continuity in the cortical substance.

Porocephalus.—The time at which porocephalus gives rise to symptoms is somewhat variable. In the majority of cases, however, the disease is present at birth or begins very early in extra-uterine life. Occasionally, the first symptoms appear after the patient has reached adult age. Unfortunately, in many of the published cases, the clinical description is very meagre. Taking into account, however, the cases only in which the beginning of the affection is noted with a fair amount of accuracy, it is found that in 55·5 per cent. the disease was congenital; in 6 per cent. the first symptoms appeared before the age of four months; in 75 per cent. before

the age of two years and in 86 per cent. before five years. In 5·5 per cent. the disease began between the ages of five and ten years, and in 5·5 per cent. of cases between the ages of ten and twenty. In 2·9 per cent. the patients were over twenty years of age, whilst, in one patient, the disease originated after he had attained the age of forty-five years. A distinction in that respect, however, must be drawn between the cases where the whole brain or a large portion of both hemispheres are absent, and those in which part of one, or a small part of both hemispheres are wanting. In the first class, the disease is almost always congenital, whilst the second class includes all the non-congenital, as well as some congenital cases.

Total or almost complete absence of the Brain.—In this first variety the symptoms call for little notice, especially as the children die at, or very soon after birth. A fact to be noted, is that the atrophy, or rather disappearance of the brain, has little or no effect on the development of the remainder of the body. These children are, as, a rule remarkably well formed and sometimes, as in the cases recorded by Tarnier[1] and Budin,[2] very heavy. Convulsions are absent, and the patients cry, take the breast, pass urine and meconium just like ordinary children, though death, often supervenes very soon after birth. One patient whose case has been reported by Billard[3] lived three days, another (Tarnier) died after six and a half hours, another (Budin) lived forty-eight hours only, but Dickson[4] saw one who lived thirteen months. One of Rokitansky's cases (reported by Kundrat[5]) lived three months, the brain being wholly absent.

Partial destruction of one hemisphere.—The disease, when limited to one or part of one hemisphere, gives rise to symptoms, confirmatory of the knowledge as to the localisations of function in the brain obtained by modern experimental investigations.

Motor symptoms are often present, as the region of the brain which regulates movements is often attacked; the sylvian artery being very frequently the seat of morbid processes. Hence, hemiplegia on the side opposite to that of

the brain-affection is a common symptom, though, strangely enough, the paralysis does not often extend to the face. This hemiplegia is often complete, but, in some cases, one limb alone (arm or leg) is affected. In the majority of cases the paralysis is accompanied by atrophy of the limb and contractures, which almost always make their appearance at some time or other of the disease.

When the whole of one hemisphere is destroyed, the paralysis is, limited to the opposite side. This, however, is not always so, as in some cases, even when there is a unilateral lesion only, the paralysis is nevertheless bilateral. Cruveilhier,[6] for instance, in his atlas, gives an account of the *post-mortem* examination of a girl, who had been completely paralysed during life. After death the *right* side of the brain only was found to be converted into a cystic cavity. Epilepsy likewise, which is a rare (7 per cent.) symptom in this disease, affects both sides as a rule, even when the brain lesion is unilateral. When the lesion is limited to the motor region of one hemisphere, although the resulting paralysis and contracture affect one side of the body, the convulsions which occur in less than 10 per cent. of these cases only, are bilateral.

Among the most interesting cases described as having occurred in adults, is one described by Poncet.[7] A soldier who died of typhoid fever, a man of a somewhat low order of intellect, fell, twelve years before death, to the bottom of a well, and part of his brain was said to have escaped through the wound. He was *left-handed* but not really paralysed, and had never suffered from epileptiform attacks. The patient died of a fever, and at the *post-mortem* Poncet found a united fracture of the left frontal bone. There was a distinct loss of cerebral substance in the region corresponding to the middle of the first frontal convolution extending into the ventricle. The middle third of the second frontal convolution was atrophied.

Voisin[8] (Leçons Cliniques) relates the history of a girl who, at the age of thirteen, suffered from right facial paralysis, left spasmodic hemiplegia, atrophy of the upper limb and arrest of intellect. At the *post-mortem*, a cyst

was found in the situation of the right ascending frontal and parietal convolutions.

In cases where the anterior part of the brain alone is affected, motor symptoms are often almost absent. Thus, Jalan de la Croix[9] gives the case of a girl, seventeen years of age, who became slightly lame on the left side when two years old. At the *post-mortem*, the area supplied by the anterior cerebral artery was found converted into a cystic cavity.

Epileptic convulsions are not frequently met with and when present, affect both sides of the body, as a rule. Unfortunately, the character of the fits has not been accurately noted in many cases, and no mention is made as to whether the attacks begin in one limb, spreading to the other, or are bilateral from the first.

The limbs are often, though by no means always, in a state of contracture and this symptom may be often absent, even in well-marked cases of para—or hemiplegia. Sometimes, though rarely, the development of the limbs on the side opposite to the brain lesion becomes arrested.

The intellectual faculties are often of a low order, many of the patients being idiots, and Heschl[10] went so far as to say that porocephalus is always accompanied by idiocy. This is far too general a statement, as the disease is not incompatible with mental qualities of a high order. A girl, fourteen years old, for instance,[11] the bearer of a cyst occupying the left side of the brain was remarkably clever, earning her living as a sorceress, somnambulist and *physician ! ! !* Kundrat[12] showed that idiocy is frequently, though not constantly, found in congenital and occasionally in non-congenital cases, even when the first symptoms of the disease make their appearance in the second or third year of life. Hence, he argues that idiocy is not due to the lesion itself, but to faulty development and to atrophic processes taking place in one hemisphere. This is no doubt true for cases in which, the patient being an idiot, small lesions only are found in the brain, but where the whole of one or both hemispheres is destroyed it is easy to understand that the intellectual faculties in such a patient must necessarily

be of a very low order. Speech is often deficient, and the patient may even be completely aphasic. In most cases, however, the difficulty of speech is the result of idiocy caused by the disease. Binswanger[13] has lately published an interesting case in which the aphasia was clearly due to destruction of the speech-centre. A man who died when forty years old, had been born with an atrophied and paralysed right upper limb and a paralysed though not atrophied right lower limb. His intellect was not of a high order, and he had never been clever enough to go to school. Though not completely aphasic, the patient had marked defects in his speech. After death, the second frontal, the two inferior thirds of the ascending parietal, the first temporal convolutions together with the lobule of the insula were missing on the left side of the brain. The interest of the case lies in the fact that, although the speech centre had been destroyed on the left side (together with neighbouring convolutions), the centre on the other side had taken up its work, though rather imperfectly.

Nystagmus and deafness are occasional but rare symptoms. Sensory troubles are seldom met with, and the writer found them mentioned in three cases only, and even in these they were very slight. The symptoms therefore resemble those of spasmodic tabes and lobar sclerosis of the brain, a point which will be again referred to later on.

The course of the disease is generally from bad to worse, the fatal termination, in a large number of cases, being due to phthisis, or to persistent epileptic convolutions. Many (37·03 per cent.) die before the age of five years, 55·5 per cent. die before the age of twenty ; 73·2 per cent. before that of forty, whilst a few live to the age of sixty, and three out of all the cases collected, lived more than sixty years.

Hydrocephalus.—Like porocephalus, the disease may be congenital or acquired.

Dr. Dickinson[14] found that out of twenty-six cases, four were congenital and sixteen began during the first six months, whilst six began at later periods up to the age of two years and two months. Dr. West[15] found that out of fifty-four cases, fifty showed symptons before the age of six months, and that four manifested signs of it at birth.

Out of one hundred and three cases collected by the writer, nine were congenital, and in ninety-three patients the disease began before the patient was six months old. This does not represent the whole truth, for in many cases of fœtal hydrocephalus the children are born dead, or destroyed during labour to save the mother, as the child is born alive in cases of slight hydrocephalus only. The manipulations to which the head is subjected during its extraction, and the faulty presentations which often occur, add to the sources of danger. Even after the body has been extracted, the bones of the head occasionally give way and extravasations of blood into the spinal canal, rupture of the neck also takes place.

The vast majority of cases of hydrocephalus, therefore, are congenital and if not actually present at birth, the disease generally makes its appearance within the first months of life. The head slowly enlarges, this increase in size being the first point to attract the parent's attention. Sometimes a fit, followed by contracture and paralysis is the first symptom or else the child cries out suddenly, is costive, restless, and shows all the signs of meningitis. Dr. West states that in twelve out of forty-five cases, fits were the first symptoms for some weeks. In six there was an attack like acute hydrocephalus and in four other instances the disease was preceded by some well-marked indications of cerebral disturbance. Squinting, nystagmus, headache are often early symptoms. As the head enlarges, the anterior fontanelle becomes more patent, the sutures widen and the head feels soft and fluctuating. In well-marked instances, the softness and thinness of the bones forming the cranial vault, the defects in the ossification, the presence of Wormian bones, together with the prominence of the fontanelles are quite characteristic. On holding a light on one side of the head, the cranium is sometimes found to be quite translucent. Fisher describes a peculiar souffle heard over hydrocephalic heads. This sign is, however, absent in the majority of cases; and, when present, is useless for diagnostic purposes. The same may be said of *Cerebral Egophony*. It is reported that in one case a cracked pot sound, and a hollow sound in another could be obtained by

percussion of the patient's head. After death, a succussion splash on striking the patient's head has been noted.

The head enlarges slowly as a rule, but this increase is not always continuous. On the contrary, there is evidence to show that the disease may remain in *statu quo* for some time and then suddenly start afresh. A hydrocephalic child may be in fair health and suddenly be seized with a bilateral or unilateral fit caused by the increased pressure in the cranial cavity. The writer has seen a patient in whom the disease made no progress for ten years. Then, without any ascertainable cause, the child was seized with attacks of giddiness, restlessness and loss of control over both sphincters. In the case of a boy seven years of age, in whom the skull was already ossified, the head suddenly increased so much in size that bones were again separated. A protrusion of the brain may also suddenly take place externally, through a bone, or through a natural opening in the skull, as the anterior fontanelle may not close till the age of sixteen or even later. In fact, it appears that the clinical phenomena ought to be divided into two stages: the *acute* period in which the patient's head increases in size quickly, the symptoms being convulsions, squinting, fever, &c., and other signs of inflammatory mischief, and secondly, the *chronic* stage, in which the patient remains in fair health, the head enlarging slowly or not at all. Either stage may of course pass into the other.

Rupture of the brain is doubtless a very rare occurrence, and as far as the writer knows, there is only one case on record in which this is supposed to have taken place. Objections might, however, be brought forward even against that case.[16] That the skull occasionally gives way is fully established.

Brown[17] and Amyott[18] both published cases in which this had taken place. Leonard H. Sedgwick[19] gives an account of a hydrocephalic boy in whom the fluid was spontaneously evacuated through the nose. After the first flow of fluid the boy improved in health for some time, but became worse later on. Twelve months after it had ceased, the flow again took place, and when it ceased, the boy remained permanently cured.

In another patient, a bloody fluid oozed through the mouth and nostrils, and when the child died, an aperture was found between the cranium and nose through the ethmoid bone. Leber,[20] in 1883, carefully observed a hydrocephalic girl in whom a continuous flow of cerebro-spinal fluid took place through the nose; though, unfortunately, she was not cured by the discharge. This flow took place only when the patient bent her head forward, but this latter fact may be accounted for by supposing that when the head was in the normal position, the fluid passed through the posterior nares. Leber further suggests that some of the similar cases described by Nettleship, Priestley, Smith, Elliotson, Baxter and Sir James Paget, were cases of hydrocephalus. Dr. Sedgwick's case was cured, and Leber's was no worse for the accident; so that the conclusion to be drawn is, that this complication is not as fatal as might be expected, especially as we know that the fluid may escape without the brain actually giving way.

Fits, as a symptom of hydrocephalus, may be unilateral or, more frequently, bilateral, and are often followed by contractures and total or partial temporary paralysis. They almost always precede an aggravation of all the symptoms and are often the herald of a fatal issue. The upper limbs of a *still-born* hydrocephalic child,[21] have been found to be flexed and rigid, the fore-arms flexed on the arms, the hands on the fore-arms, and the fingers into the palms of the hands, the biceps-muscles being strongly retracted, and standing out prominently at the elbow joints.

Partial or general contractures are frequently met with in very young children, however, and like fits, generally mean a speedy death. Complete paralyses are of very rare occurrence, and the patients, though often unable to stand, are usually, when in the recumbent position, able to move their limbs freely. Temporary hemiplegia, paraplegia even, have been noticed to follow convulsions. The eyes often look outwards and upwards, sometimes to the right and at other times to the left, varying in direction from day to day in the same patient. More rarely, they are directed downwards, or one eye only is affected. In one patient the

squint remained, even after the primary disease had been cured by appropriate treatment.

Bouchut[22] was one of the first to draw attention to the state of the *fundus oculi* in this disease. In 1865, speaking of the diagnosis between chronic hydrocephalus and rickets, he states that, as the first-named disease progresses, the following changes take place in the fundus :—

1. Great vascularisation of the papilla of the retina with dilatation of the veins, the latter, however, preserving their natural colour.
2. Augmentation of the veins of the retina.
3. Partial or complete serous infiltration of the papilla.
4. Atrophy of retina and vessels.
5. Almost complete atrophy of the optic nerve.

Similar changes have been described since, by Allbutt,[23] Swanzy,[24] Leber[25] and others ; but these appearances are by no means always present in the *fundus oculi*, and there are but few cases on record in which such changes have been noticed. The blindness sometimes disappears when the child is cured of the primary disease. The writer has examined several patients without discovering any defects in their appreciation of colours, or in their field of vision.

Anosmia has been met with in some patients.

The state of the pulse and respiration does not seem to have attracted sufficient notice. In the cases the writer has seen, the respiration was normal in character and frequency, the pulse varying between 70 and 90, but in three cases the acceleration of pulse after the least excitement was much more marked than is normally the case, as the beats rapidly went up to 120 per minute and kept on at that rate for some time. In two out of fourteen patients, the writer found the patellar reflexes distinctly exaggerated. The other reflexes were normal.

The digestive system rarely gives rise to symptoms, though vomiting is sometimes one of the earliest signs which may persist through life. Costiveness has also been noted by several authors. Polyuria was noticed in one boy. Sleepiness and loss of control over the sphincters are occasional symptoms.

A young child suffering from hydrocephalus presents a characteristic appearance as a rule. When the patient is at rest in bed, the head, if very large, lies helplessly on the pillow and the sufferer is often quite unable to move it in any direction. The veins covering the head are enlarged, the skin is glazed and shiny, the cranium looking as if it was almost on the point of bursting, the scanty hair and the comparatively small triangular face together with the big head giving the patient an aged and worn appearance. The squint and perpetual nystagmus as well as the exophthalmos before noticed add to the strangeness of the child's appearance. In some cases, on pressure being applied to the anterior fontanelle, the patient falls into a kind of stupor which lasts as long as the pressure is maintained.

The child sometimes remains in one position for hours, but may be suddenly convulsed, sick, cry out or give other signs of agitation and pain. The limbs move about freely enough as a rule and sensation is normal, but the body, even if fairly large at birth, soon becomes puny and cachectic, especially should the head become much larger. Should the patient survive and reach the age of one or two years, he may learn to walk. The head is often too heavy for the child, and one hydrocephalic patient told the writer that the weight of his head often caused him to fall forwards. On examining hydrocephalic patients the writer has in three cases seen symmetrically placed on both frontal eminences a number of small scars evidently due to former falls. After a time, however, the patients may be able to walk well, but in slight cases of hydrocephalus even the body often remains undersized and weak. Thus an hydrocephalic girl, sixteen years old, only measured 74 centimeters in height.[26]

The intellectual faculties, even when the head is large, are often of a higher standard than would have been expected. Cardinal, the patient whose case has been placed on record by Bright, was fairly intelligent, and a patient whom the writer had an opportunity of seeing, earned his living by going about the streets singing comic songs and making stump speeches, by no means devoid of readiness and

wit. Bouchut[27] states that he knew two men above fifty, who, though markedly hydrocephalic, had intellectual talents of no mean order. The writer is acquainted with two similar cases. The first is a man fifty-three years old, and the other, a remarkably clever medical man, thirty years old. Others, however, even with smaller heads, are almost idiotic, for the state of the intellect does not altogether depend on the size of the head. Some never learn to read or write, appear to be insensible to kindness and often unable to speak. They cannot walk, look utterly idiotic, sit in one position with the saliva dribbling out of the half-open mouth, not taking any interest in things going on about them, hardly better, intellectually, than the lower animals. These cases are chiefly met with in lunatic asylums.

There is one fact which seems to have escaped the notice of writers on this subject, namely, the relation of hydrocephalus to deafmutism. In the writer's opinion the two conditions often go together. Through the kindness of M. Hugentobler, the writer was able to examine the fifty-one deaf and dumb mutes in his establishment at Villeurbanne, and found that thirteen of the inmates had very large heads, but that four of them, *i.e.*, about 8 per cent., were unmistakably hydrocephalic. These children varied in age from nine to twelve, were all fairly well-built, though three of them were distinctly undersized. They were quite deaf, not hearing the shrillest whistle close to their ears, but had all learnt to speak, though one of them had acquired the faculty of speech with a little more difficulty than the other pupils in the establishment. Physical examination of their eyes, ears and bodies, revealed nothing abnormal, except the large head above mentioned. In no case were there any signs of rickets, but one of them had syphilitic teeth and a suggestive scar in the back. One of these children was very clever, watching the speaker's lips with great attention and speaking French fluently and correctly. Two of them showed exaggeration of the patellar reflexes and the reaction of the pupils to light and accommodation was sluggish in all. Their digestive functions were normal, but M. Hugentobler informed the writer that some of them would go on eating

voraciously until told to leave off, though the same authority finds this to be frequently the case in deaf children. Their genital organs were well formed and none of them showed any inclination towards masturbation.

These children had never been ill and no cause, except hereditary syphilis in one case, could be discovered for their affliction.

Blindness and anosmia have been described, but the other cranial nerves are normal as a rule, though in one case a difficulty in swallowing liquids is noted.[28]

Unlike the majority of patients with congenital intellectual deficiency, the sexual appetite in hydrocephalic children appears to be normal, though in two cases an early precocity is noted in that respect.[29]

It is impossible to say what is the average duration of life in patients afflicted with hydrocephalus. With a very large head even (see case of Cardinal, reported by Bright), the patient may live to reach adult age, and the writer is acquainted with one man, aged thirty-nine, whose head is so large that he dare not show himself in the streets, who though undersized and puny, is in very good health and able to walk and talk. Bouchut knew of two cases above fifty years of age; and one seventy-one years of age, has been reported by Gölis.[30] When old these patients often become maniacal, melancholic or simply idiotic and end their days in asylums. The prognosis is, on the whole, very bad.

The cases recorded in medical literature, in which the patients have lived to adult age, are few, and the writer has been struck with the paucity of *post-mortem* records of adults. Nearly all such records refer to children under two years of age. In fact, on reading through the notes of the cases in medical literature, the author found that 68 per cent. died before the age of two years, and 80 per cent. before the age of ten years. Although these statements are, of course, only approximate, yet they give an idea of the duration of life in hydrocephalus. Death may be due to various causes, *e.g.*, repeated convulsions, marasmus, meningitis, or to intercurrent diseases, or to bursting of the head.

Dr. Blache stated that he had never seen a hydro-

cephalic child survive an attack of measles, and, as a rule, these children offer little resistance to intercurrent diseases. In some cases the head seems to have become less distended just before death and in Dr. Baxter's case the external protrusion through the frontal bone shrank visibly just before the fatal termination.

III.

The conclusion arrived at was that in the large majority of cases of hydrocephalus the disease is congenital, though the influence of sex, heredity, &c., had not been mentioned.

Strangely enough, porocephalus is more frequently met with in females than males, whereas the reverse is the case in hydrocephalus. The latter fact has been noted by Morgagni.[31] Out of the cases collected by the writer: 59 per cent. of cases of porocephalus occurred in females, and 41 per cent. occurred in males. Whereas of hydrocephalic patients, 75 per cent. belonged to the male, and only 25 per cent. to the female sex.

Heredity has no influence in the production of porocephalus, as might well be expected, but it appears to play some part in the production of hydrocephalus. Out of twelve children[32] of one hydrocephalic patient, only one ininherited his complaint, but one hydrocephalic woman[33] had four children all markedly afflicted with the same complaint. The father and brother of the hydrocephalic girl, whose case has been recorded by Leber,[34] had unusually large heads, but considering that only very few patients afflicted with this disease reach the age of maturity, it would be strange indeed if the affection was frequently hereditary.

The disease occasionally runs in families, or the same woman may have several hydrocephalic children in succession. In one instance, it is stated that, of two sisters, each gave birth to hydrocephalic children.

Dr. Hale White[35] gives the following account of the brothers and sisters of four hydrocephalic children:

1. Male child. Three hydrocephalic children are dead. One is hydrocephalic now.

2. Female child. Elder sister had an unusually large head.

3. Male child. Of eight brothers and sisters, one had an unusually large head.

4. Of four children from one mother, two have died of chronic hydrocephalus, one is now suffering from it, whilst one has escaped. The first three were born by instruments, but the last was born naturally.

G. de Grandmont[36] found that in one family two (three?) children out of six were hydrocephalic; and J. P. Frank records the case of a woman who gave birth in succession to seven children all afflicted with this disease. Similar cases have been noted by Underwood,[37] Armstrong,[38] and also by Simpson.[39] In Gölis's[40] work will be found the history of one woman who had six still-born hydrocephalic children. The seventh child lived eighteen months, and the two last were born healthy. According to Fritsch,[41] when several children of one mother are born with this affection, they mostly belong to the microcephalic variety. This is not the opinion the writer has formed from his reading, for he has been unable to find any family histories in which this had happened.

Syphilis has occasionally some influence in the production of the disease. In porocephalus, the writer has found a history of syphilis in 4 per cent. of all cases only, but in so-called chronic hydrocephalus, he has found hereditary syphilis mentioned in 20 per cent. of the cases examined by him, or reported in medical literature. Poulet, however, gives the proportion as 10 per cent. only. Osiander,[42] Haase,[43] Rayer,[44] Gros,[45] de Méric,[46] Hutchinson,[47] Lancereaux,[48] Laschkewitsch[49] and quite recently again Sandoz,[50] have all drawn attention to syphilis as a cause of hydrocephalus. Haase[51] reports the case of a woman who had a chancre during the first half of her pregnancy, the husband being evidently syphilitic. She had three still-born children, but the fourth child was hydrocephalic, hemiplegic, with purple spots disseminated on the skin and died when six months old. The other children born in later years were evidently syphilitic.

De Méric[52] observed, on several occasions, an enlargement

of the head and peculiar rotatory movements in the eyes of syphilitic children.

Lancereaux[53] was told of a woman whose husband had contracted syphilis some years before marriage, and who gave birth to several hydrocephalic children; whilst Hutchinson[54] has noticed the co-existence of the two affections. According to Laschkewitsch,[55] syphilis may determine in the brain lesions of an inflammatory type, namely, inflammation of the ependyma, with consecutive inflammation of the ventricular liquid (?); this state being accompanied by other syphilitic lesions. This condition is characterised by continuous and progressive headache, nausea and vomiting, the symptoms being more marked when the patient moves about. The intellect becomes dulled, the pupils are dilated though contractile, and hyperæmia of the *fundus oculi* is noticeable with the ophthalmoscope. The pulse is unaltered, and there is no fever.

Georges Sandoz[56] believes in the existence of a syphilitic hydrocephalus, which may be congenital or develop in the first few months of extra uterine life. According to this author it is most probably caused by inflammation of the ventricular ependyma and plexuses, the morbid process being due to hereditary syphilis.

If syphilis be a cause of hydrocephalus it is difficult to understand why the latter disease is not more common. According to most authors, hydrocephalus occurs once in 3000 confinements, hence syphilis (syphilis being present in one out of five cases of hydrocephalus), acts as a cause of hydrocephalus about once in 15000 confinements. Now, syphilis is a common disease and it might be expected that, if it stood in any causal relation to the other disease, hydrocephalus would be more commonly met with. But, if in one out of five cases of hydrocephalus a distinct syphilitic history is found, it may fairly be argued that the first is sometimes the cause of the second, especially as to these cases might certainly be added some whose parents did not know they had suffered from that infectious disease, and some who wilfully conceal the fact of their having been thus afflicted.

In any case, syphilis is not the only cause of the disease, for the disease is described as occurring in animals (calves, goats, sheep, &c.), which are never affected by syphilis.

In 23 per cent. of all cases the writer found a history of tubercle in the families of hydrocephalic children, but never in the hydrocephalic fœtus, and in 4 per cent. only of the children who lived for some time after birth.

Poulet[57] states that hydrocephalus occurs chiefly among poor people, so that tubercle may possibly act as a cause, by weakening the health of the father and mother.

The coexistence of syphilis and tubercle in hydrocephalus, both being distinctly infective processes, raises the question as to whether, in some cases at least, the disease is not due to an infective process which ran its course in utero. We know now, that after the human body has successfully resisted an infective process, the evil effects of the latter do not always come to an end, even when the disease has to all appearances been mastered. During the course of scarlet fever, for instance, or after it has subsided, the urine may show that the kidneys have been affected by the infectious process, and though, in the majority of patients, the disease either ends in speedy death or perfect cure yet in some, long after all traces of the primary disease have disappeared, the morbid action set up in the kidneys still continues, may gradually add to the patient's danger, or even cause a fatal termination, although the real infective process came to an end long before the patient's death.

Syphilis is another example. A patient who has had syphilis, may become ataxic after all traces of the primary disease have left him. The sclerosis taking place in the posterior columns of his spinal cord is not a syphilitic process, for it resists the action of specific remedies, it differs from it in its anatomical characteristics and in its clinical course. Although the connection between syphilis and locomotor ataxy is, thanks to Dr. Gowers' work, established on a firm basis, locomotor ataxy, although often due to syphilis, is not a syphilitic process. Numerous examples (*e.g.*, in the ætiology of cerebro-spinal sclerosis, some forms of heart disease, &c., &c.) might be brought forward, to

show that an infectious disorder may set up after a time, a morbid process in some organs; this process, although not infective itself, being nevertheless due, in the first instance, to the primary infective disease. In the same manner, an infective process present in the father, mother, fœtus, or newly-born child, might, even after the cure of the primary disease, possibly give rise to inflammation of the lining membrane of the ventricles of the fœtus.

The frequent occurrence of hydramnios in the mothers of hydrocephalic children, together with the lesions found in the fœtal vessels, also point to the disease being occasionally caused by syphilis.

Rickets coexisted with hydrocephalus in 13 per cent. of all cases, and has often been mentioned as a cause. Vinsoneau[58] especially lays great stress on the frequency with which the two diseases are combined. Considering, however, the wretched hygienic conditions in which most of these children are placed, the number of congenital cases and the frequent absence of all traces of rickets, the influence of the latter as a cause can only be very small.

Alcoholism, consanguineous marriages, advanced age of father and mother, epilepsy in the father, have occasionally been invoked as causes, but it is impossible to draw any certain conclusions as to their ætiological value. The forceps have been incriminated by some on the *post hoc ergo propter hoc* principle. The writer has been unable to find any confirmatory evidence on that point.

Dareste[59] has lately shown, that by subjecting eggs to somewhat rough usage, the embryo is often malformed in various ways. (Abnormal development of the brain, encephalocele, &c.) Blows, &c., to the mother might possibly cause the same in man, but the writer has only heard of two cases where this was supposed to have been the cause of hydrocephalus, and they neither of them bear close investigation. In 8 per cent. of all cases of porocephalus there was a distinct history of a blow, the onset of the disease being consecutive to the traumatism. Violence, therefore, must be admitted as a cause. In hydrocephalus, external violence has been reported in about 2 per cent. of cases only, but has been noted by as eminent an observer as Dr. West.

Cretinism, as a cause of hydrocephalus, has been mentioned in almost all text-books. The author cannot say why this idea should have gained ground, for he has been quite unable to find any confirmatory evidence. The only case in favour of that opinion is that published by Stoltz[60] in which a cretin gave birth to a hydrocephalic fœtus. Poulet made inquiries on the subject in Switzerland, and was unable to gain any confirmatory evidence as to any causal relations between rickets and hydrocephalus.

Syphilis, hereditary tubercle, blows and deficient hygiene, are therefore the only causes known to favour the development of the disease.

The ætiology of porocephalus calls for a few special remarks. Many causes have been invoked to account for origin of the porocephalus.[61]

Breschet[62] has attributed most cases to an arrest of development. In a great many *post-mortem* examinations, however, traces of old hæmorrhages, disease of vessels, &c., have been noted. In these cases arrest of development as a cause of the disease must be excluded. Moreover, the remainder of the brain presents, as a rule, no other malformations. In a few cases, however, the presence of hare-lip, fissure of the face, malformation of other convolutions seem to point to some early fault in development.

Encephalitis has been assumed by some to act as a cause, but it appears to the writer without there being any definite facts to support that theory. The pathological anatomy of the disease reveals the fact that the lesion is often exactly limited to a vascular area and changes (obliteration) of the vessels of that part, produced through thrombosis or embolism, are frequently found at the autopsy.

It would serve no useful purpose to discuss whether the changes in the vessels are primary phenomena, or secondary to changes in the placenta and heart, as Kundrat supposes. It is sufficient for our purpose to know that the appearances have been described by many, and among others by Duret, Budin, Klebs, Kundrat,[63] and that the cyst-like formation in the brain is sometimes due to disease of the vessels of the cortex.

Where the cortex is nearly intact, the cyst is often formed by the dilatation of one of the horns of one ventricle. The pathological cavity may communicate with the ventricle, or may be cut off from it by a band, the result of a previous inflammation. Blows seem to have been the primary cause of the disease in a few cases.

Porocephalus is therefore the result of two distinct processes :—

A.—Disease originating in cortex (blows, hæmorrhage, encephalitis, &c. A form generally accompanied by loss of cerebral substance).

B.—Disease originating in ventricles.

There are no real points of difference therefore between cases of porocephalus belonging to the latter category and hydrocephalus. The liquid accumulates in the brain in both, but whereas in the first it distends one ventricle or part of one ventricle only, in the latter it distends both ventricles though not always in an equal degree.

IV.

The brain of a patient suffering from hydrocephalus is distended and the cerebral substance compressed owing to the increased quantity of fluid present in the ventricle. As the accumulation of fluid increases, the pressure on the surface of the encephalon rises pari passu, owing to the cerebro-spinal fluid pressing on the lymphatics and on the walls of the veins of the arachnoid membrane.

It has been proved experimentally, that when the intracranial pressure is increased, the venous flow is arrested first; but that if the pressure rises still more, the flow in the capillaries comes to a standstill, and the anæmia of the brain substance thus produced gives rise to symptoms of compression of the brain.

Leyden[64] has proved by experiment, that an increased pressure of the cerebro-spinal fluid in animals gives rise to restlessness, groans, screams, irregular respiration, together with marked slowing of the pulse.

Epileptiform attacks with opisthotonos, extension of the

hinder extremities, deep and irregular respirations, followed by coma and flaccidity of the limbs, dilatation of the pupils, increase in the rapidity of the pulse (300 per minute or more), vomiting, loss of control over the sphincters follow in quick succession. A very gradual increase of pressure does not produce these effects.

Acute symptoms similar to those are present often enough in hydrocephalus, though they are sometimes absent from first to last; the intra-cranial pressure, owing to the yielding of the skull, being less than might be expected.

When convulsions and other symptoms occur, it may be assumed that they are due to a sudden augmentation of intra-cranial pressure. The presence of paralyses, contractures, &c., calls for no special explanation, and the occurrence of optic neuritis must be accounted for in the same manner as it is in other cases of increased intra-cranial pressure.

Most of the symptoms, as well as the disturbances in intellect, may possibly be caused or made permanent by the changes which, in one case, the writer has proved to have existed in the cortex of the brain.

The central canal having been found closed in a number of *post-mortem* examinations, it has been suggested by many that the distension of the skull and the symptoms of the disease were due, solely and simply, to the obstruction to the flow of the cerebro-spinal fluid and to the increased intracranial pressure produced by the slow and gradual distension of the cranial cavity. Many facts, however anatomical, clinical and pathological are opposed to this view.

The ventricular cavities are not closed sacks, communicating by one foramen only, with the cerebro-spinal canal and arachnoid cavity; for since the classical investigations of Key and Retzius and others, it is an admitted fact that there are many points of communication between the ventricles and the outer surface of the brain. The fourth ventricle communicates with the sub-arachnoid spaces, firstly, by the foramen of Magendie; secondly, by the foramen of Bochdalek and the *aperturæ ventriculi quarti*

laterales, first described by Luschka and rediscovered by Axel Key and Retzius.

The ventricles of the brain communicate with the fourth ventricle through the aqueduct of Sylvius, but Key and Retzius have found that, by injecting coloured substances under low pressure into the sub-arachnoid spaces of the spinal cord, a direct communication between them and the hemispheres of the brain could be traced in both animals and human beings; this coloured substance distributing itself into the velum interpositum, and also into the choroid plexus. The lateral ventricles always contained some amount of free fluid which may penetrate through the aqueduct of Sylvius into the fourth ventricle, and even into the central medullary canal. These communications certainly suffice for the evacuation of the fluid normally present, even after the closure of Magendie's foramen. But whether all these openings, including Magendie's, would be sufficient when, through inflammation, a large quantity of fluid is poured into the cerebro-spinal cavity is very doubtful; for we know that large serous membranes like the peritoneum, are often quite unable to absorb the inflammatory fluid poured into their cavities.

The pathological liquid found in the ventricles is not normal cerebro-spinal fluid, but differs from it in the fact that it contains far more albumen, and a larger quantity of certain salts, is of higher specific gravity, may be turbid and contain pus-corpuscles, is more highly-coloured and resembles in some respects the product of an inflammatory liquid.

Though an obstruction has sometimes been proved to exist, yet in many *post-mortem* records there is no mention of any impediment to the flow of liquid being discovered. Magendie[65] himself reported cases in which the foramen called after him was not only open, but was even larger than natural. In a remarkable case of very large hydrocephalus published by Gaucher,[66] this competent observer distinctly states that the fluid ran with the greatest ease through the foramen Magendie into the arachnoid cavity. In Mr. Hutchinson's[67] patient, the hydrocephalus communicated by

a large canal with the spinal bifida, in the lumbar region. It may also be mentioned that, in the horse, the foramen is normally closed, and that in man, Axel Key and Retzius have found it closed without its giving rise to hydrocephalus.

It is difficult to understand why, when there is no obstruction present, the fluid should not, following the laws of gravitation, descend into the spinal canal. The writer can offer no explanation of it, except by calling attention to the fact that, in other cases of accumulation of fluid in the head, the liquid does not always find its way into the spinal canal.

The progress of the disease is not always a gradual one, as it certainly would be supposing the clinical phenomena to be due merely to a slow and gradual accumulation of fluid, but the morbid process may remain quiescent for years, may stop at any time and may even be cured, if convulsions giddiness, and other symptoms do not point to the supervention of further mischief in the cranial cavity. Thickening of the ependyma by interrupting the flow in the veins of Galen and by obstructing the central canal may possibly give rise to some retention of cerebro-spinal fluid, and this retained liquid may itself act as a further cause of irritation; the beneficial effects of tapping being explained by the removal of one source of irritation.

We know, for instance, that some forms of ascites are cured, or improved for a time, by a judicious tapping. If, however, the cause of disease is a chronic inflammation of the membrane lining the cavities of the ventricles, and if, as in most chronic processes, sudden exacerbations sometimes take place, then the character of the fluid, the occasional sudden onset of the disease, the convulsions, headache, vomiting &c. find a ready explanation. If due to mere continuous passive accumulation of fluid, it is difficult to understand why the disease should occasionally stop in its progress, or even be cured. If the cause is an inflammatory process (one resembling for instance, that giving rise to a hydrocele) it is easy to see that, when the morbid action has run its course, or when treatment has successfully interfered, the patient may remain in *statu quo* or permanently recover.

Tubercle and syphilis have already been alluded to as important ætiological factors. Now tubercular patients are, of course, subject to remissions and to exacerbations, but this is especially the case with syphilis. A syphilitic patient may remain in perfect health for some time, without showing any signs of the disease, but under the influence of bodily or mental ill-health, the syphilitic symptoms suddenly reappear. This is exactly what happens in hydrocephalus, and the writer assumes that, in some cases at least, the sudden exacerbations may be due to a new outbreak of the syphilitic virus.

The presence of fluid, and the symptoms, in the writer's opinion, are not due to mere passive accumulations of fluid in the ventricles, but are the result of an occasional acute, but usually chronic inflammation of the membrane lining the ventricle. The obstruction is secondary to the primary inflammation and although possibly intensifying, is not necessary to the production of the disease.

If hydrocephalus be compared with other congenital defects of the cerebro-spinal axis, several points of resemblance with diseases which, at first sight, appear to be very dissimilar, may be found. A true myelocele, as Virchow has shown, is always due to the dilatation of the central canal of the spinal cord. If this dilatation be slight, the cord is almost normal in appearance; but should more liquid accumulate, the spinal cord ruptures, the bony walls of the spine atrophy through pressure and the tumour protrudes externally; that is, posteriorly, laterally, or even anteriorly. If, for the sake of simplicity, we assume the brain to be a tube surrounded by walls thicker than those of the spinal cord, the same process will produce similar effects, but the *post-mortem* appearances will vary according to the time at which the morbid action has begun. Let us suppose, that in early fœtal life, whilst the skull is almost unossified, a ventricle or part of one ventricle slowly dilates. The cyst thus formed, whether it be cut off from the remainder of the ventricle or not, gradually increases in size and perforates the skull, not protruding through a fontanelle as a rule but through a bone, and an encephalocele is the result. In

another patient, the brain has been partly or almost completely destroyed through disease in early fœtal life, the convolutions are absent, or only traces of them exist, the encephalon is represented merely by a large cyst, and the fœtus presents the characteristics of hydrocephalus or porocephalus. In still another patient, this dilatation has begun late in fœtal or early in independent life, *i.e.*, when the brain is already formed, and the bones developed to some extent.

The effusion of liquid has taken place into a closed cavity which expands by slow degrees, giving rise to a large hydrocephalus. Like a spinabifida and like an encephalocele, hydrocephalus may become latent, *i.e.*, not increase in size, or may suddenly enlarge, so much so that the walls may suddenly give way.

Spinabifida is frequently associated with hydrocephalus and hydro-encephalocele. In fact, in some patients in whom that disease begins in the neck, there may be a hernia of the brain and spinal cord through the sanæ foramen. In others (*e.g.*, in Mr. Hutchinson's) the whole cerebro-spinal system is affected. It is only fair to assume that the same pathological process which produces hydrocephalus in the brain, gives rise to a myelocele when it attacks the spinal cord or to both, when both the brain and spinal cord are affected.

The various dilatations, therefore, which occur in the cerebro-spinal central canal, the writer has arranged under the following heads :—

1. Dilatation of the central canal of the spinal cord giving rise to a myelocele. This may burst during fœtal life and form a spinabifida occulta.

2. Dilatation of one ventricle or one horn of it, forming a porocephalus. This may protrude through the skull and is then called an encephalocele ; or it may burst after working its way through the skull, and give rise in early fœtal life to an encephalous monster.

3. Dilatation or destruction of both hemispheres in early fœtal life, with premature ossification of the skull. (Porocephalus.)

4. Dilatation of both hemispheres late in fœtal or early in independent life. (Chronic hydrocephalus.)

V.

The difficulty in the diagnosis of hydrocephalus depends largely on the extent of the lesion and on the size of the patient's head. In cases where the whole head is enlarged to a slight extent only, the disease may be taken for one of hypertrophy of the brain, a mistake which, according to some authors, is by no means uncommon. It must be remembered, however, that the probabilities are always in favour of its being a case of hydrocephalus; and secondly, that symptoms such as paralysis, contractures, fits, optic neuritis, point in the same direction. Intellectual defects, moreover, are rare in hypertrophy of the encephalon, but frequently present in hydrocephalus. So little, however, is known of the former disease, that a slightly marked case would present the greatest difficulties.

The fontanelles and the whole cranium are enlarged in the rickety child, just as they are in hydrocephalus, but always to a far less extent. The presence of a well-marked chain of Wormian bones, the gaping of sutures are in favour of the disease being hydrocephalus. Lastly, the rickety deformities in other bones, with a head only slightly enlarged, point to the disease being due to rickets only.

In a well-marked case the diagnosis is easy, as there is no other disease in which the head attains the enormous dimensions which have been recorded. If the disease be limited to part of one hemisphere or to a few convolutions —when the head is not enlarged—the diagnosis becomes extremely difficult and it is almost impossible to form an opinion in such cases. The only difference between hydrocephalus and the disease due to chronic meningo-encephalitis, &c., is the absence of hemiplegia in the latter.

It differs from atrophic sclerosis of the brain, only in the fact that the latter is frequently not congenital, and more often secondary to infectious diseases. (Richardière).[68] Lastly it has to be distinguished from the diseases giving rise

to hemiplegia in children and especially from the one-sided paralysis due to vascular lesions. It is needless to enter into the diagnosis between it and cerebral tumour, or tubercular meningitis, the symptoms being entirely different.

Vascular lesions of the cerebrum are excessively rare in childhood, although hæmorrhages have been described by a few authors. The writer fails to see how they could be distinguished from porocephalus either in their mode of onset or clinical course. The same applies to the endarteritis due to syphilis, although the latter might possibly be distinguished through the effects of treatment. Lastly, we must not forget that vascular changes may themselves produce porocephalus.

In conclusion, therefore, although the diagnosis of the well-marked forms is quite easy, that of the slighter forms is in almost all cases almost impossible.

TREATMENT.

Porocephalus admits of no treatment either preventive or otherwise, but the treatment of chronic bilateral hydrocephalus must be divided into two parts, namely, surgical and medical.

The surgical procedures which have been advocated are :—

1. Tapping.
2. Pressure and tapping combined.
3. Injection of iodine.
4. The introduction of a seton.
5. Permanent antiseptic drainage.

West, in 1845, collected all the cases treated by puncture (56 in number) and found that the cures amounted to a little over 28 per cent. By adding Dr. West's cases to those the author has collected (69), the number of successful cases after tapping amounts to 21·75 per cent. only. Hydrocephalus is an almost hopeless disease, however, and any procedure therefore which cures one in five cases and which, as a rule, does no harm (for in the writer's cases only three seem to have actually suffered from the effects of the puncture) may conscientiously be tried. König[69] advises the use of a trocar previously disinfected by heat and carbolic acid.

An assistant exerting some pressure with his hands on the head, the instrument is to be introduced at a spot about 265 centimetres distant from the sagittal suture and is to be pushed, avoiding the longitudinal sinuses, right into the ventricle. It is as well to draw the skin over the seat of puncture beforehand so as to form a valve-like opening. Thompson[70] drew off 10oz. and some more escaped afterwards. Bruns[71] advises to draw off 2-4oz. at a time, but should the fluid flow readily more may be drawn off with advantage. The ease with which the flow takes place may therefore serve to some extent as a guide to the amount of fluid which is to be drawn off.

Malgaigne[72] lays down the following rules :—

The operation may be performed,

1. In a patient three or four months old, even when the hydrocephalus appears not to be increasing.

2. In a patient over four months old, if the head be not already ossified, and if the hydrocephalus increases and constitutes a danger to the life of the individual.

Bruns advises the operation only if the distension is considerable, if the fontanelles and sutures are widely open, if the bones of the head are free and mobile, if the child is well nourished, not paralysed and is physically and intellectually as advanced as a normal child of the same age, and lastly if the hydrocephalus be continually increasing in size.

It is impossible to draw up a code of rules to be applied to all cases. Some cases get well spontaneously, so that if the disease is not actually progressing, it is only right to see what nature will do, but if, after using drugs, mercury, iodide of potassium, &c., &c., the child steadily gets worse, the head ought to be tapped. The presence of paralysis need not be a counter indication, for cases have been recorded in which the child, though blind and paralysed, recovered sight and power of walking after the cure of the primary disease.[73] If one tapping be not sufficient, the operation may be repeated successfully as in Thompson's case, though it is but seldom that, after one has failed, repeated operations have any good effect.

Compression has been warmly advocated by many,

especially at the beginning and the middle of this century; but has deservedly fallen into disuse again. Rivierius in 1656 mentioned pressure as a cure for hydrocephalus. Sir Gilbert Blane in 1821 claimed to have cured hydrocephalus n that way, and in 1823 Barnard[74] speaks of six cures thus obtained in his own practice, and mentions that Dr. Engleman was successful in ten patients. Trousseau[75] for some time advocated pressure, but having seen a patient die in consequence of it, condemned the practice.

The development of the effusion, says Trousseau, being limited at its superior part of the skull owing to the compression[76] "the base had given way just as it gives way when, in order to separate the bones of the skull, anatomists fill it with water and beans which, swelling up, produce the disarticulation." Dr. Dickinson[77] lost one case through sloughing of the scalp during compression but Phillips[78] reports that (on a boy whose head was almost on the point of bursting) he tried the application of pressure; the child recovering and being alive and well ten years afterwards. He mentions that the same method was also successful on another of his patients.

Injection of iodine has been tried several times, notably by Winn, Barnard[79] and Tournesko, but has as far as the writer is aware only been successful once in the case reported by Tournesko.

A seton has been used always with a fatal result. Haven[80] in one patient tried permanent antiseptic drainage but with a fatal result, and electricity is reported to have cured one patient.

Medically, hydriodate of potash and the iodides of potassium and iron have been used without success. Bromide of potassium has been advocated by Brunton[81] who has cured a case by its use.

If, as the writer supposes, the disease is frequently associated with the syphilitic diathesis the use of mercury is distinctly indicated and there is some evidence that the latter drug is useful, Gölis[82] and Gowers[83] have both cured cases in that way, and Umpleby[84] in the *British Medical Journal* of 1871 has recorded a remarkable case of cure by its use.

The child in question was twenty-two months old, had been blind for six months, and its head measured 27 inches in circumference. Dr. Umpleby ordered :—

℞.

Hyd. c. Cret.			
Rhei.	}	aa	gr. ii.
Sod. Carb.			

or,

℞.

Hyd. c. Cret.		gr. ii.
Pulv. Ipec. c. Op....		gr. i.

according to the child's condition.

Three months afterwards the child was able to walk, had, at three years of age, recovered its sight perfectly, and was, except for its large head, quite well. Dr. Umpleby further states that his father cured a child in the same way. In a case of progressive hydrocephalus the writer thinks that mercurial treatment might be tried first with advantage, having recourse if it failed to tapping.

It is my pleasant duty to thank my friend, Prof. Pierret, for suggesting this subject to me, as a thesis, for much advice and for kindly allowing me to make use of the vast pathological material of Bron Asylum.

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Reviews and Notices of Books.

A Text-book of Mental Diseases: with special reference to the Pathological Aspects of Insanity. By BEVAN LEWIS, L.R.C.P.(Lond.); M.R.C.S.(Eng.) Charles Griffin & Co., London, 1889.

THIS is in many respects the most important book on Insanity extant. As one lays it down an inclination arises to compare it with the works of Bucknill and Tuke, Griesinger and Maudsley; but reflection shows this to be impossible, as the condition of scientific knowledge of the anatomy, physiology, and pathology of the nervous system differed at the various periods at which each was written. Bevan Lewis has the advantage of being the youngest man on the list, and of having worked in an atmosphere cleared by his predecessors of much of the mists of tradition which hung around the subject. All the works cited must live as successive enunciations of the philosophical methods of the study of insanity, and each marks a mile on the road. Lewis has added another mile, or perhaps two. He has had the path greatly cleared for him, and has had brilliant light thrown on it, afforded by the results of recent investigations. But his work is by no means solely based on the observations of others; he has worked with his own hands, and has taken a prominent place amongst the men who have done so much of late years to open up the secrets of the nervous system. His arduous labours have been productive of many important results, and it is curious to note how meagre has been the recognition of his work in Germany, and even in this country; we know of no man who has done so much solid work, who can append to his name such a small list of honorary distinctions.

In one respect this book is unique:—it begins with an elaborate exposition of the anatomy of the brain and spinal cord. If we mistake not, this is the first treatise on insanity which deals in a thorough manner with the anatomy of the organ implicated. It may be thought by some, that for the practical purposes of the work the first six chapters are unnecessarily diffuse, and that a

more general description would have sufficed. But the author probably thought it well to provide the student of nervous disease with ready access to full information, holding that as such knowledge becomes more generally diffused so the probability of the explanation of obscure clinical phenomena will be increased. Lewis writes on Meynert's lines, and occasionally in Meynert's obscure style. Not having availed himself of the recent results of the developmental method of tracing fibre, certain of the descriptions of tracts in the medulla (*e.g.*, the fillet and olive), and of the relations of nerves to nuclei (notably the auditory) are not entirely satisfactory. The two chapters on the structure of the cerebral cortex, however, are in every respect admirable. A well-known investigator remarked to us lately that they contain the very best description of these regions in this or any other language, and this opinion we unhesitatingly endorse. They should be at the fingers' ends of every man who attempts to treat brain disease, for, without full and intimate knowledge of the anatomy of the convolutions, no one has a right to advance even a theory of the genesis and progress of pathological processes occurring in them. Whoever does so is in the position of the man who, unacquainted with the structure of the kidney, treats of "dropsy." This applies specially to the vascular and lymphatic systems of the cortex and adjacent white matter; and of both a clear and vivid description is afforded. The implication of these systems is at the root of most disturbances of consciousness, and the author cannot be too highly complimented on the manner in which he has demonstrated their arrangement. It is open to question whether he does not attach too much importance to the action of the "lymph connective" tissue cells. This, however, is a question which must remain *sub judice* for some time to come. And, again, it is fair to take exception to his statement that the channels between the brain substance and the hyaline sheath "are not the lymph spaces proper," but that they communicate with the epicerebral space of His. His holds that this is a lymph space, and it is, therefore, somewhat difficult to reconcile the two opinions, as it is not easy to realise the existence of non-lymphatic channels debouching into a lymphatic cavity. Our own opinion is strongly against the existence of any epicerebral lymph-space, and we hold that the channels are mere involutions of the naked surface of the brain.

This section concludes with an all too short and too restricted reference to physiology. In a work which deals so fully with

anatomy it might have been expected that the functions of tissue would have received more detailed consideration. It is true that physiological observations of the highest interest are scattered throughout the book, but its general purpose would have been better served had the subject been treated of in more concentrated form. Physiology should have been systematised equally with anatomy. The significance borne by the nucleus in the autonomy of the nerve-cell is worthy of all consideration; but are there not other matters deserving of prior attention. The question of vaso-motor action in relation to the cerebral vessels is not considered; and we have to seek in vain for any detailed observations on the cause of sleep, on cerebral rotation, and on other matters which would have materially assisted the author and reader, both in the clinical and pathological sections.

The clinical section is the result of the experience of four thousand cases occurring in the hospital, which of all others, has done most in promoting the scientific study of insanity; it bears the mark of thorough hospital work, the application of many scientific experimental aids to diagnosis, and of careful recording. The observations on reaction-time, the condition of the blood, and electrical stimulation are especially valuable. The chapters on epileptic insanity, general paralysis, and alcoholic and senile insanity may be regarded as perfect, as least as much so as the present state of knowledge permits. They contain much original matter.

It seems inevitable that authors on insanity must make an effort to analyse disturbances of consciousness from a purely psychological standpoint; and accordingly Lewis, in the chapters on states of depression, stupor, and exaltation, projects a scheme of morbid psychology highly ingenious and seductive, but in our opinion having no bearing on the pathological position, the elucidation of which is the main object of the book. The whole matter is hypothetical, and it would be by no means difficult for a man of like ability belonging to another school of philosophy, to devise a system equally seductive, and equally useless to the physician. We hold with Griesinger that "the entrance to psychiatric medicine is not through the dark portal of metaphysics," and that nervous disease at large, has not been in any way developed "from a specially philosophico-psychological point of view." The psychological method has done much to retard the objective study of the many diseased conditions of which insanity is a symptom. But in this one respect Lewis has followed the beaten track: he "walks the chambers of the (insane) brain,"

and finds no difficulty in tracing out the sequence of psychological events from the incidence of simple morbid depression of feeling, to the most complicated phenomena of delusion, morbid impulse, and mental decadence. The thesis is interesting, here and there vague, especially when attempts are made to collate physiology with mental phenomena, but not harmless: for it leads the author away from consideration of the main issue—the physical causes of implication of the substrata of consciousness. Here he fails us. Early in the chapter on depression he assumes "a state of *cerebral torpor* in the physical substrata of *object-consciousness*, and a state of *cerebral irritation* in the substrata of *subject consciousness*;"[1] but he fails to offer even a theory as to what produces the torpor or the irritation. The reader is left to infer, in the absence of any further information, that a psychological nexus only exists between the cause and the symptom without the intervention of any direct material changes in the cortical tissues.

What the student desires to learn is in what the primary irritation consists, and how it acts in producing "quantitative and qualitative variations in the nutritive functions of the nervous centres arising from direct disturbance of the blood current in one group of so called varieties of melancholia, and in another induced in the nervous tissues primarily."[2] (Page 129). The matter, to say the least, is not outside theory. The condition is, so to speak, idiopathic; it begins primarily in the brain, and is due to its over-excitation, whether we regard the assigned causes to be "adequate" or "trivial." Is it not then worth while to enquire whether the action of the nervous tissues may not produce disturbance of blood supply directly or reflexly. Here we must again remark on the absence of all consideration of vaso-constrictor or vaso-dilator nervous supply to the vessels of the encephale. Are we to infer that the author agrees with certain physiologists that such systems do not extend to the brain? If so, it should have been stated. This is not the place to express our own views on the subject; but we demur to the opinion that "the very earliest signs preceding genuine pathological depression are really the symptoms of cerebral anæmia and nervous exhaustion." This we hold to be a traditional assumption, based on psychological

[1] Whilst copying this passage we must enter a protest against the double-leading of words to which importance is attached; it fails to effect the purpose intended by reason of constant repetition, and mars the look of the page. It rather suggests a fulminating psychosis.

[2] The sentence between inverted commas is paraphrased from the text.

considerations, and that physiological, clinical, and pathological data point to the early signs of idiopathic melancholia being symptoms of hyperæmia of the superior convolutions, followed, perhaps, in the later stages by anæmia induced by secondary structural changes in or around the vessels. Lewis, again, alludes to toxæmia as a "qualitative variation of the blood-plasma" inducing depression; but he does not enquire where the change arises, nor alludes to the possibility of the brain supplying its own poison. Where he fails us is in ignoring the extremely difficult question of the patho-genesis of idiopathic disease of the convolutions, produced by over-exercise of function, and symptomatised by mania, melancholia and dementia; he does not go far enough back in his pathology, and takes no account of the physiological work of Gaskell, Foster, and Roy, and little of that of Mosso and Duret, which might have served to add importance to his observations on the vascular unity of the brain in the last section of the book. It is interesting to observe that when we reach such subjects as epilepsy and general paralysis, where the author feels the ground of pathology more firmly under his feet, psychology is discarded, and true clinical work only occupies the page. Were it not that this book is pitched high, the above criticism would be inadmissible.

Conspicuous by its absence is all reference to syphilis as a factor in the production of degeneration of nervous tissue, implying the author's disbelief in its action. In such cases it is as valuable for the reader to know what the author disbelieves as what he believes.

The last section treats almost entirely of morbid anatomy. It is of the highest excellence; no one in Great Britain, save Lewis, could have written it, and we doubt whether on the continent any individual observer could have produced such a series of chapters. Interspersed with the details of morbid histology we find pages characterised by a philosophic pathology, the outcome of deep thought and study, which in subsequent editions will probably be further elaborated. It is hopeless to offer a digest of the contents of this section; the morbid changes found affecting each individual structure are described with a minuteneness which to some may appear almost painful, but which is absolutely necessary for the purposes of any student who desires to seek below the surface. The illustrations are delicately executed, and represent with great accuracy the various degenerations. Those who have not examined morbid, or for that matter, healthy brains, by the freezing and aniline-

black staining process, may be inclined to regard many of the pictures as somewhat diagramatic. This is not so, each illustration delineates with exactitude delicate structures which by this method are readily demonstrable.

There are, however, certain points which demand explanation, and others calling for criticism. In the chapter, for instance, on general pathology and morbid anatomy, in which the appearances presented in all forms of insanity are discussed, certain lesions are ascribed to inflammatory action. The pathological anantomy of chronic alcoholism, epileptic insanity and general paralysis are considered separately, so that we must infer that the general chapter treats of the morbid appearances presented in other forms of insanity, except where the distinction is specially noted. Certain of the changes in bone and membranes are held to be evidence of inflammatory action of old standing; but in the clinical section we find no evidence that the author regards inflammation as a factor in the production of mental symptoms, general paralysis excepted. Where then, and in what forms does it occur? If it is of old standing it must have existed very early in the disease, probably it was the disease itself. If it was not so why did it occur at a later period? "The *pia mater* is abnormally thickened in fully 48 per cent. of those dying insane, partly from fibrinous exudates which have organised, partly from plastic lymph, and often from an œdematous swollen condition of the conjoined soft membranes," &c. This is a fair picture of "bygone inflammatory action," and we must ask the author when and under what circumstances was it existent. Again, in dealing with atrophy of the convolutions, Lewis assumes that all excess of cerebro-spinal fluid is compensatory. No doubt it is so in the very great majority of old standing cases, and in small local atrophies; but in fatal recent cases of acute idiopathic insanity and general paralysis, accumulations of fluid are found which, had they occurred in the subjects of basal meningitis would have been held to be the immediate result of inflammation. Lewis holds strongly that general paralysis is an inflammatory disease, in which opinion we thoroughly concur; that being so, why should it be denied exudation, one of the first and most important of the products of inflammation? which being poured out in quantities larger than the lymphatics and veins can carry off, presses on the convolutions and produces atrophy. We hold that the question is definitely settled by the phenomena presented in an

early case of general paralysis, on which the operation of trephining was performed at our instance a year ago. A disc was removed from the left parietal bone and the brain did not bulge into the hole appreciably. The same operation was performed at the corresponding spot on the opposite side, and the *dura mater* bulged so much as to occupy the trephine hole. The patient died eight months after the operation from pneumonia: on examination, a large accumulation of fluid was found in the side which had bulged, and there was a corresponding atrophy of the convolutions; on the other side there was little or no fluid, and no atrophy. If the fluid had been compensatory the membrane would not have been forced into the hole. The same has been observed by Dr. Claye Shaw in two cases in which he caused the operation to be performed. Holding the compensatory theory, Lewis is precluded from considering the important influence which is exercised by fluid pressure in the production of symptoms.

The pathological novelty of the book is the theory of the action of the large Deiters, or connective tissue-cells. Lewis holds that the "Lymph-connective" elements of the brain, or "scavenger cells," as he calls them, take a very active part in the processes of disease affecting the nervous centres. In his own words:—"The delicate system of lymph-connective elements permeating in the normal state, the whole of the cerebral mass of white and grey substance, takes a more active share in the pathogenesis of mental decadence than any other; and the more the question is investigated, the greater the importance, we feel convinced, will be attached to these elements in the processes of disease as affecting the nervous centres. Their physiological indications are clear; they are the *scavengers of the brain;* and the evidence obtainable renders it now incontrovertible that they are liable to excessive and rapid development under certain morbid conditions affecting cerebral nutrition and repair . . . . . Whatever leads to increased waste of cerebral neurine, whenever structure disintegration is slowly proceeding, either in nerve cell or fibre; whenever accumulation of *débris* occurs from disease of the vascular tracts, then we invariably note an augmented activity, registered in these scavenger elements of the brain. That their activity is in direct ratio to the functional activity of the essential neurine tissue we think there can be no doubt; nor that with each accession of the nerve-tide they are stimulated to increased activity in the removal of the products of waste and the plasma effused from the vessels.

In healthy states, however, they never assume the hypertrophied form, the deep staining, the coarse fibrillation, the rapid multiplication, and the evidence of obvious inter-cellular digestion, which are readily observed in pathological states." This hypertrophy of the processes being distributed between the nerve elements and surrounding the vascular walls, replaces the delicate neuroglia, and as the cells undergo further alteration they produce a fully formed felt-like material, which destroys the lateral poles of the ganglionic cells, and by ultimate contraction interferes with the permeability of the vessels. Whilst agreeing with the author to a considerable extent, we cannot help thinking that he places undue importance on the action of the scavenger cells on the one hand, and too little on the other; and that we must guard against the theory that there is associated with them in the brain a pathological process different from what occurs in other organs. It is generally accepted that connective tissue cells act as phagocytes in most parts of the body, whether in health or disease. In disease a prominent example is afforded by the connective tissue cells of the lung taking up foreign matter and removing it into the lymph stream, or encapsulating particles. In the early stages of certain cases of catarrhal pneumonia epithelium may be considerably proliferated with but slight apparent increase of connective tissue, which only becomes very marked on the condition becoming more or less chronic. This seems to indicate that the more embryonic (and therefore less easily demonstrable) the connective cells are, the more active is their phagocyte action, and the more they work to remove effete material; only becoming, so to speak, hypertrophied after long subjection to irritation. In other cases the connective tissue becomes rapidly, it may be almost said primarily involved, and the pathological consequences are more serious. We believe the same types of change occur in the brain. In such diseases as general paralysis and chronic alcoholism, the increase of connective tissue resembles that of the latter class of catarrhal pneumonias; in certain other forms of insanity, that of the former, the cell not losing its embryonic character, and therefore acting strongly as a phagocyte, and procurer of restoration to normal conditions. In the diseases mentioned, probably on account of the irritation acting more directly on the spider-cells they proliferate so rapidly as to destroy the protoplasmic poles of the cortical cells by contracting, interfere with the potency of vessels, and by injuring tissues which cannot be reproduced, render recovery

impossible. The strong probability is that in idiopathic and other insanities these "lymph-connective" cells play an important phagocyte rôle, for in that condition there is a large amount of effete matter to be removed, and which, as shown by the large percentage of recoveries, is removed. No one will contend that this is the only agency at work in getting rid of *débris* and devitalised tissue, but there are strong reasons for believing that its influence is considerable. We are inclined to believe that the large easily demonstrable scavenger cells of Lewis are cells which have passed through the more active phagocyte stage, and which, although by no means powerless, have suffered from the over-exercise of function. The reticulum formed by their processes interferes with the healthy relations of tissues, and, as Lewis points out, tends to destroy the association system of ganglionic cell-poles. This reticulum, in fact, constitutes the invariable sclerosis of general paralysis, and the occasional sclerosis found in other forms of insanity. It is a curious fact that where sclerosis is found to have existed, exaltation of feeling has, in a large proportion of cases, been manifested during one period or another of the course of the disease. In general paralysis it is almost invariably present. It is the only definite mental symptom which can be associated with a particular structural change. Can no one offer a theory of the relation of symptoms to lesion?

Lewis asserts that this felting of the processes of the Deiter's cells does not occur in the epileptic insane. Here we differ. In the very last case of this condition which we examined, the proliferation was remarkably well marked, particularly around the larger vessels, and what may be of some importance to notice, the bodies of the large ganglionic cells in the neighbourhood were to all appearance healthy, except that their poles were specially well marked; so much so as to suggest that they were undergoing analogous changes to those of the neuroglia cells. The cell nuclei presented slightly the vacuolation mentioned in the work; but is not this appearance nearly as common in other forms as in epileptic insanity?

Eulogy is thrown away on this book: it speaks for itself in every page; the veriest tyro must see, although he may not appreciate all its merits; and there are very few workers in the same field of enquiry who will not be obliged to admit that Lewis has carried his observations in morbid histology beyond their own range. But much remains untouched even by this book, which

may be said to contain all that is at present known of the morbid appearances presented in the insane. The first new point to be worked out is the locus and period of incidence of structural changes. It is almost a waste of time for the student now-a-days to examine by any very elaborate method the brains of the subjects of old standing insanity; he must apply himself to recent and acute cases which have manifested mental symptoms in any form—coma, delirium, insanity—and to those who have died from head injury. Imitative experiment may also assist. Our belief, founded on an as yet very limited number of observations, is that many changes which have been regarded as secondary are in point of fact primary, and that they are produced with extraordinary rapidity.

JOHN BATTY TUKE.

Injuries and Diseases of Nerves and their Surgical Treatment. By ANTHONY A. BOWLBY, F.R.C.S. 1 vol., 8vo, pp. 510. London: J. and A. Churchill, 1889.

THIS is the work of a skilled microscopist and accurate clinical observer, and it abounds with facts of the greatest interest and importance. It may be spoken of in terms of highest praise, for after very careful reading we believe that it will take a place beside the classical treatises of Létievant and Weir Mitchell. It marks indeed a distinct advance on the works of these two authors, for not only has Mr. Bowlby been able to avail himself of a literature upon the subject which has much increased since the issue of their books, but microscopical methods have also much improved, and more therefore can be learned of the pathological changes which are the result of nerve injury. The book moreover is not a mere compilation of the work of others; it is full of original matter, based upon the careful observation of an unusually large number of cases in the practice of the Surgeons of St. Bartholomew's Hospital during a period of nine years. And what makes the contents so valuable is the fact that many of the cases have been pursued and observed with unrelenting watchfulness for a long time, so that something might be known as to the ultimate results of treatment in cases

of suture of divided nerves. Nearly half the book is indeed devoted to the subject of nerve suture, and is one long plea in favour of this plan of treatment, supported by the strongest evidence as to its usefulness and good. We commend the lessons therein to all surgeons.

It is our intention in this notice to refer to those parts of the book in which Mr. Bowlby has something new to tell us, or in which his opinions differ from those of previous writers.

As to the regeneration of nerves the importance is pointed out of not relying too much upon the results of experiments on animals, for there seems to be considerable variety in the exact mode both of de- and re-generation in different animals. It is very necessary therefore to trace the process in man. He confirms the observation of Hayem that new nerve elements are to be found in the bulbous end of a nerve divided in amputation, but he goes further than this and gives three cases in which regeneration of the peripheral ends of nerves had occurred after section in continuity independently of union with the upper portion. It is strange that any regeneration should occur without reunion with the nutritive centre, but as to the fact the author has no doubt. This however has to be noted that the regenerative effort is probably short-lived, and that unless union be accomplished degeneration again sets in and may go on to complete atrophy. No stronger argument could be adduced in favour of nerve suture, for it is obvious that connection with the nutritive centre is essential for the continuance and perfection of the process which may have been begun in and by the nerve itself.

Discussing the question of primary union he regards it as quite possible without any antecedent degeneration, for the complete return of sensation and motion is a sufficient proof of it in at least two cases. It is of course very rare, and anything which prevents healing by first intention must oppose the primary union of nerves. The author expresses his firm conviction that the commonly accepted doctrine that *partial* section or wound of a nerve trunk is more dangerous and more likely to lead to complications than complete section is quite erroneous. His experience is moreover opposed to the well-known dictum of Weir Mitchell that "the capacity of nerves to remake their lost parts is almost invincible," for the records of surgery in untreated cases in man tell a very different tale. Nerves whose ends are

separated show no irresistible tendency to unite. If they unite at all they do so in a most imperfect manner and frequently there is no sign of repair.

The chapter on the trophic changes induced by injury is rich in material. He doubts whether glossy skin, as stated by Mitchell, is "never present without burning pain," or causalgia.

Several instances are recorded, with illustrations, of changes in the nails, and in more than one the interesting fact that all the nails of a hand were involved even though the nerve supply of some fingers only had been directly affected. Thus after division of the median nerve, not only may the nails of the index, and middle fingers, and of the thumb show trophic changes, but those of the ring and little fingers may also frequently be affected at the same time and in a similar way. However much may be the median supply to the ring finger it is certain that the little finger has none from this nerve, and the suggestion is offered that the alteration in the nail of the little finger is of a reflex nature.

Arthritic lesions of a mild and chronic kind are in the author's experience amongst the most common results of nerve injury. In from one to six weeks the joints, those of the fingers more especially, will be found stiff and sometimes swollen, painful and tender. Finally there is tolerably firm fibrous anchylosis. This may become permanent in exceptional instances.

He does not agree with Charcot that muscular atrophy occurs most commonly after injuries which are prone to induce a neuritis to which therefore the atrophy is presumably due, and he has no hesitation in saying that in by far the larger number of cases which he has himself seen, and in which there have been trophic lesions more or less marked, the nerve section has been complete and there has been no reason whatever to suspect any neuritis. He believes therefore in the direct trophic influence of the nerves upon the tissues, and that in all probability the same fibres are capable of conveying the different impulses which are generated in the centres.

Supplementary sensations have always been of great and unexplained interest after nerve section. The extent to which anæsthesia is present requires an immense amount of care to estimate, and many recorded observations are probably inaccurate. Nevertheless there are cases in which the retention of a certain amount of sensation has been unquestionably noted after section. In Mr. Bowlby's own experience such supplementary

sensation appears to be most marked immediately after the section and subsequently disappears. One thing is quite certain that a simple return of sensation does not afford sufficient proof of new nerve formation, and that in order to settle this, there must be evidence of improved nutrition; trophic lesions must have passed away; the atrophied muscles must have resumed their normal bulk, their contractility, and reaction to faradism.

Not less remarkable than the trophic changes and the affections both of muscles and joints are the deformities which result, and here the author has something very definite to tell us. He points out that there is no active contraction, as is commonly supposed, in the atrophying muscles and that although they do shrink and grow smaller in every diameter there is not sufficient force in the process to cause actual deformity. The whole secret of it lies in the fact that the muscle shrinks only so long as it is unopposed. If the origin and insertion of a muscle be unduly approximated during the process of atrophy, the muscle remains in that position when the trophic changes have ceased, not because the origin and insertion have been drawn together by such changes, but because the atrophy found the muscle in a certain position and there fixed it. Examples are given in support of this view.

Turning to the practical results of nerve suture we find the author's experience leads him to take a decidedly sanguine view. Although union by first intention is not a thing to be expected, nevertheless the probability is that function will be restored. And here comes the further important conclusion which has been drawn from watching the cases over a long period of time. No definite answer can be given to the question how long it will be before recovery ensues in any given case. It may be weeks months or years. Considerable improvement will probably occur within a year, but the record of cases shows that a much longer time than this may be necessary for the complete restoration of function. No case, he says, should be given up as hopeless until the lapse of at least two or three years from the date of suture, whether primary or secondary, and not even then if any improvement is in progress. Our own experience is in full accord with this opinion. Moreover the nerves of young persons regenerate both more surely and more rapidly than do the nerves of the old, and the prognosis of suture of the musculo-spiral nerve seems better than in the case of the median and other nerves.

We have thus chosen, and have put forward almost in his own

words the views of the author on several points of special interest, and the same might have been done in connexion with other parts of the book, wherein he deals with many topics, to wit: reflex paralysis, the various complications of nerve injuries, the uses of electricity, the respective merits of neurotomy, neurectomy and nerve stretching. Enough however has been said to show the character of the work. The bibliographies which it contains, and excellent plates, very much enhance its value, and we cannot doubt that the book will have a large number of readers, who will derive from it both profit and enjoyment, that it will advance our knowledge of an important branch of pathology, and largely tend to the better and more successful treatment of some very serious injuries.

HERBERT W. PAGE.

Abstracts of British and Foreign Journals.

Syphilis.—Insanity and General Paralysis, By Dr. A. Morel-Levallée (*Gazette des Hopitaux*, Oct. 19th, 1889).

The author maintains that there are morbid conditions of brain, due to certain poisons, of which the syphilitic is one, which deserve the name of pseudo-general paralyses, because 1stly, they differ from general paralysis in pathological anatomy; 2ndly, they differ from it also in some points of the evolution of the disease, more especially in the frequency of remissions, and even in their curability, but he admits the difficulty of differentiating the pseudo from the true general paralysis. Unfortunately the name syphilitic-pseudo, general paralysis, has been applied to two series of clinical facts having no pathological connection with one another, dependent upon (*a*) separate syphilomata, (*b*) a diffused periencephalitis attributed rightly or wrongly to syphilis. The author enters into a history of opinion as to the influence of syphilis in producing mental disease and specially general paralysis; he believes that syphilis is an important cause of general paralysis, but whether it should be regarded as a predisposing or an exciting cause, pathological anatomy does not permit us to say.

Essential Myopathies. By Dr. Raymond (*Gazette des Hopitaux*, October 6th, 1888).

The author considers as an essential myopathy, every amyotrophy in which the muscular fibre seems primarily diseased, and presents an alteration which appears not to be referable to nervous trouble. But it is difficult to exclude the influence of functional disturbance of the nervous system.

Essential amyotrophies may be divided into two groups. 1. Local atrophies confined to a single group of muscles, or even to a single muscle. 2. Progressive primary amyotrophy. Those in the first group are not due to any material lesion of the nervous system, but probably depend upon some functional disturbance of the trophic centres, which, however, is purely hypothetical. In

the second group the intervention of the nervous system is much less probable, and we may believe that the atrophy is due to a primary change in the muscular fibre.

The type facio-scapulo-humeral appears usually in childhood, and first attacks the facial muscles. Besides its peculiarity in picking out the muscles implied by its name, it is distinguished by important characteristics. Fibrillary contractions are absent. Electrical contractility is modified quantitatively, but not qualitatively; there is no reaction of degeneration. The tendon reflexes are preserved. Muscular shortening occurs. The affection progresses slowly; the muscles of the special mechanisms are not involved. The disease is hereditary, and may be a family one. The physiognomy is characteristic. In the single case in which an autopsy was made, absolute integrity of the nervous system was found. The juvenile form of progressive muscular atrophy of Erb has many resemblances to this type, but differs in the absence of facial implication, and in the presence of accompanying hypertrophy of certain muscles, often, however, followed by atrophy. There are close resemblances between this type and pseudo-hypertrophic paralysis, and they may be regarded as varieties of the same disease, which probably includes also the hereditary form of progressive muscular atrophy of Leyden. The fact that these varieties are linked together by transition forms, is a strong argument that they are really one disease.

Beyond heredity no cause is known. No lesion of the central or of the peripheral nervous system has been discovered by the most careful examination, but many observers believe that the disease depends upon a functional disturbance of the trophic centres.

The Mental Troubles of Old Age. By Dr. ROUILLARD (*Gazette des Hopitaux*, July 13th, 1889).

In speaking of premature senility, the writer points out that it is sometimes susceptible of amendment, and must be distinguished from true senility, which always goes on to a fatal termination. Still more necessary is it to distinguish the pseudo-senility of young men. In $\frac{9}{10}$ of the cases premature senility must be attributed to atheroma. Apart from this morbid condition we find it only in melancholics and cachectics.

The first symptom of senile dementia is failure of memory. The memory is destroyed little by little, layer by layer. The failure at first seems slight, and is most marked for recent events. The memory for names, and especially for proper names, is the

first to go. The other faculties undergo an analagous deterioration. The will is almost destroyed. The moral and affective faculties are not less impaired; there is exaggerated emotionalism.

As complications of senile dementia are mentioned—delirious senile dementia, *i.e.*, senile dementia accompanied by delusions and hallucinations—delusions of persecution. These differ from the same delusions in delusional insanity in not being coordinated, and in being multiple, changeable and fugitive. Hallucinations present the same characters as the delusions. They are not always disagreeable. Visual hallucinations are the most common. Illusions, misrepresentations of sense impressions are still more common. Melancholic and hypochondriacal delusions; of these the physical condition is the most powerful factor. Patients may appear melancholic who are really not so. Religious delusions; their presence makes the prognosis more unfavourable; erotic ideas; ideas of grandeur.

In old men indecent acts are usually prodromata of dementia they often occur before there is obvious mental failure.

Morbid Anatomy. — Two facts dominate senile dementia arterial atheroma and brain atrophy. Atrophy affects the cerebrum more than the other parts of the brain, and especially the left hemisphere and the temporo-parietal region. The writer does not share in the views of Luys, who regards loss of weight as much more important than atheroma.

Schneider on Nasal Reflex Epilepsy (*Berl. Klin. Woch.*, No. 43, 1889).

The author relates six cases which were cured by treatment of the nasal abnormality.

I.—Male, æt. fourteen years, without hereditary taint; epileptic since the age of five years. Fits occurred nightly while falling asleep or shortly after commencement of sleep. Often an asthmatic attack immediately preceded a fit. Both nares were almost occluded by polypoid swellings; a large mucous polypus hung from an unciform exostosis of the septum. Touching the inferior turbinate swellings of either side educed sibilant bronchial râles and cough, with great anxiety of facial expression, followed in the course of half an hour by a severe fit. The patient has had no epileptic or asthmatic seizure since the morbid growths were removed in November, 1885.

II.—Female, æt. twenty-four, no heredity; epileptic from the time of puberty; subject to vertigo and migraine. For some

years received benefit from bromide. The origin of the epilepsy was then (July 1885) accidentally disclosed by the occurrence of a severe fit which followed an attack of sneezing brought on by looking towards the sun. The nasal mucous membrane was found to be greatly tumefied; reflex cough was excited by contact of various points. On the right inferior turbinate was a spot, irritation of which caused mydriasis of the same side and exclamation of feeling ill. The mucous membrane was brought into a healthy condition by cauterisations, since which the patient has been free from epilepsy, vertigo, and headache.

III.—Absence of fits since the tumefied nasal lining was reduced by the cautery two years ago.

IV.—A strongly built man, free from heredity, æt. thirty-five, in 1885 suffered from frequent dry cough, dyspnœa, paroxysms of asthma with vertigo and "flying heats," also from occasional convulsive attacks with loss of consciousness. Rhinoscopy shewed a small exostosis surmounted by a soft tumour, the size of a large pea, on the right lowest turbinate. When this tumour was touched patient began to cough, complained of irritation beneath the sternum, and pain in the back; sibilant dyspnœa, extreme anxiety of expression, and dilatation of right pupil ensued; patient started up with a scream, stood rigid for a moment, then fell in a fit. Immediately after removal of the soft tumour (an angioma in which were many nerve filaments) the patient expressed complete relief from all his symptoms. Was immune for a year; occasional vertigo then developed and recurrence of the soft growth was discovered. This and the exotosis were extirpated. So far, perfect recovery has resulted.

V.—Male, æt. forty, no heredity; has been under treatment for two-and-a-half years on account of various nervous troubles; had suffered from epilepsy for some months; complained of severe asthmatic paroxysms. During even slight mental excitement convulsive tic, blepharospasm, and salaam convulsion appeared. Several polypi were found attached to each inferior turbinate bone; since they were destroyed (two and three quarter years ago), all the convulsive and asthmatic seizures have been absent.

VI.—Female, æt. seventy-two, began to suffer from mimic convulsion, nystagmus, and severe blepharospasm after the occurrence of a nasal catarrh. Attacks of unilateral clonic wryneck became established and gradually developed into *le haut mal*. On the right lowest turbinate there was a large soft tumour, cauterization of which (two years ago) was followed by an immediate and entire cessation of all the nervous symptoms.

In one case of long-standing epilepsy with nasal polypi this treatment has failed.

Schæfer on Railway-spine (*Ibid*, No. 43).

A case is cited to show that the neuropsychosis designated as above may result from mental shock alone. The patient, a strong temperate man, while driving his engine round a curve suddenly observed the three red lights of a train that was standing on the same line of rails fifty metres in front of him. He stopped his train in time to prevent a collision, but during the moments of danger he suffered violent mental agitation, producing uncontrollable tremor of his legs. In the course of a few days he utterly broke down and rapidly emaciated; gait was trailing and laborious; speech difficult and stammering; hands trembling; tongue showed coarse and fibrillar tremor; pulse was quickened to 104; reflexes exaggerated in a moderate degree; Romberg's symptom present, also much general muscular enfeeblement. Disposition excitable and melancholic; eyes red from weeping; patient sought solitude, displayed great fear, had frequent vertiginous attacks and constant severe headache. Memory greatly impaired. Sleep much disturbed by terrifying dreams. Very marked diminution of cutaneous sensibility; a needle prick sufficient to draw blood was scarcely felt as a mere contact sensation. Subjectively the legs felt cold, numbed and paralysed, and there were frequent flashes of light before the eyes. Pupils equal and re-acted well. Anorexia; taste disordered. Involuntary micturition.

E. Birt.